A DISSERTATION ON

A STUDY ON CLINICAL AND ECHOCARDIOGRAPHIC EVALUATION OF NEONATAL CARDIAC MURMURS AND THEIR FOLLOW UP AT 6 WEEKS OF AGE.

Submitted to

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M.D DEGREE IN PAEDIATRICS

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DECLARATION

I solemnly declare that this dissertation "**A STUDY ON CLINICAL AND ECHOCARDIOGRAPHIC EVALUATION OF NEONATAL CARDIAC MURMURS AND THEIR FOLLOW UP AT 6 WEEKS OF AGE**" was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem-636030 under the guidance and supervision of **Prof. Dr. T.S. SUNDARARAJAN M.D.,DCH.** Professor and HOD of paediatrics **& Prof. Dr. R.SIVAGAMASUNDARI M.D., DCH,** Professor of Paediatrics, Govt. Mohan Kumaramangalam Medical College and Hospital Salem-636030.

This dissertation is submitted to the Tamil Nadu Dr.M.G.R Medical University, in partial fulfillment of the University Rules and Regulation for the award of **M.D BRANCH VII PAEDIATRICS.**

PLACE: SALEM **(DR.R.SARANYA)**

DATE:

CERTIFICATE

This is to certify that this dissertation entitled "**A STUDY ON CLINICAL AND ECHOCARDIOGRAPHIC EVALUATION OF NEONATAL CARDIAC MURMURS AND THEIR FOLLOW UP AT 6 WEEKS OF AGE**" is a bonafide work done by **DR.R.SARANYA** in **M.D BRANCH VII PAEDIATRICS** at Government Mohan Kumaramangalam Medical College Hospital, Salem-636030, to be submitted to the Tamil Nadu Dr.M.G.R Medical University, in partial fulfillment of the University Rules and Regulations for the award of M.D BRANCH VII PAEDIATRICS under my supervision and guidance, during the academic period from May 2010 to April 2013.

Prof.R.VALLINAYAGAM, MD.,
DEAN
Govt. Mohan Kumaramangalam
Medical College and Hospital
Salem-636030

Prof.T.S.SUNDARARAJAN, M ,D.,D.C.H
Professor and HOD of pediatrics
Govt. Mohan Kumaramangaam
Medical College and Hospital
Salem- 636030

CERTIFICATE BY THE GUIDE

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Prof. **R.SIVAGAMASUNDHARI M.D., DCH.**

Professor of Paediatrics.

Govt. Mohan Kumaramangalam

Medical College and Hospital

Salem-636 030.

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LIST OF ABBREVIATIONS

1. ASD- Atrial septal defect
2. AGA- Appropriate for gestational age
3. AS- Aortic stenosis
4. CHD- Congenital heart disease
5. CHF- Congestive heart failure
6. CoA- Coarctation of Aorta
7. ECG- Electrocardiogram
8. ECHO- Echocardiography
9. HLHS- Hypoplastic left heart syndrome.
10. IVC- Inferior vena cava
11. LA- Left atrium
12. LGA- Large for gestational age
13. LV- Left ventricle
14. LVH- Left ventricular hypertrophy
15. PA/IVS- Pulmonary atresia with intact interventricular septum
16. PFO- Patent foramen ovale
17. PA- Pulmonary artery
18. PVR- Pulmonary vascular resistance
19. PGE_1- Prostaglandin E_1
20. PAPVR- Partial anomalous pulmonary venous return
21. PHT – Pulmonary hypertension
22. RA- Right atrium
23. RV- Right ventricle
24. SGA- Small for gestational age
25. SVC- Superior vena cava
26. TAPVR- Total anomalous pulmonary venous return

27. TGV- Transposition of great vessels
28. TOF- Tetralogy of fallot
29. TR- Tricuspid Regurgitation
30. VSD- Ventricular septal defect.

LIST OF CONTENTS

ABSTRACT

A STUDY ON CLINICAL AND ECHOCARDIOGRAPHIC EVALUATION OF NEONATAL CARDIAC MURMURS AND THEIR FOLLOW UP AT 6 WEEKS OF AGE

Background: Congenital heart disease (CHD) is one of the most common congenital malformations. Presence of cardiac murmur could be a warning sign of serious CHD. This study is done to emphasize the meticulous examination of the neonates for murmur and early referral for diagnosis and interventional therapy.

Objectives: a) To determine the presence or absence of heart disease in neonates with cardiac murmurs. ***b)*** To identify clinical characteristics which differentiate pathological murmur from functional murmur. ***c)*** To assess the reliability of routine clinical neonatal evaluation in diagnosing CHD. ***d)*** Follow up of babies with detected lesion at 6 weeks of age to know about the evaluation of murmur.

Methodology: Hospital based prospective study was conducted for a period of 12 months. All term neonates delivered in our hospital with cardiac murmurs were taken up for study. Detailed history was taken and associated clinical features noted. Chest X-ray and ECG were taken. Following which all neonates underwent echocardiography. Parents of babies with detected abnormality were counseled. These babies were followed up at 6 weeks of age for persistence of murmur and CHD.

Results: Among 4116 deliveries, 98 neonates with cardiac murmur were taken up for study. The percentage of having CHD among neonates with murmur was 53.06%. The incidence of heart disease in our hospital was 12.6%. VSD was the commonest defect. Incidence of CHD is higher in neonate of consanguineous parents. It is possible to make clinical diagnosis of murmur as innocent or pathological based on associated clinical features, X-ray and ECG abnormalities.

Conclusion: It is important to evaluate all neonatal cardiac murmurs before they become symptomatic. Early diagnosis aids in early referral and appropriate intervention. These reduce morbidity and mortality .By evaluation of neonates based only on murmurs few CHD can be missed.

Keywords: Neonate, Cardiac murmur, congenital heart disease (CHD), Echocardiography.

INTRODUCTION

Congenital heart disease is one of the most common congenital malformations. It remains the major cause of death in babies with congenital malformations. The common clinical features with which neonates of CHD present are heart murmur, respiratory distress, cyanosis and CCF. They may also have abnormal chest x-ray and ECG findings.

Murmur is suggestive of structural heart disease. Sometimes it could be functional also. It may disappear after appearing. Detection of murmur should prompt early referral for investigation, diagnosis, intervention, and appropriate family re assurance. This would reduce morbidity and mortality in newborn with congenital heart disease.

Congenital heart disease occurs in approximately 0.8% of live births. The incidence is higher in stillborns (3-4%), spontaneous abortuses (10-25%) and premature infants (2% excluding PDA).[1]

With advances in both palliative and corrective surgery, the number of children with CHD surviving to adulthood has increased dramatically. Critical CHD requiring surgery or catheter based intervention occurs in 25 percent of those with CHD.[2]

About 55% of babies with CHD may be missed as many would be asymptomatic at birth. Hence a more careful and detailed examination of

a neonate is essential. Clinical presentation of neonates may mimic other conditions such as sepsis and therefore a high degree of suspicion is necessary to diagnose CHD.

This study is done to emphasize the significance of meticulous clinical examination of neonate and role of murmurs in evaluation of heart disease.

AIM OF THE STUDY

- ***To determine the presence or absence of heart disease in neonates with cardiac murmur.***
- ***To identify clinical characteristics which differentiate pathological murmur from functional murmur.***
- ***To assess the reliability of routine neonatal clinical evaluation in diagnosing CHD.***
- ***Follow up of babies with detected lesion at 6 weeks of age to know about the evolution of the murmur.***

REVIEW OF LITERATURE

Mehrdad Mirzharahimi MD et al[3]

In this study, 2928 newborns were screened for cardiac murmurs. Of them 91(3.1%) neonates had murmur (prevalence: 31 cases per 1000 live births). 47(51.6%) neonates were found to have CHD. In 48.4% of neonates the murmur was innocent. VSD was the most common abnormality (17.6%). PDA was the second common (11%).Around 50% of neonates with cardiac murmurs has CHD. No significant relation between birth weight and CHD was detected (p=0.4). No significant relation between gestational age and CHD was detected (p=0.8)

Gregory R.Samson et al[4]

Persistent murmur at or greater than 48 hours after birth is strongly in favor of structural cardiac disease. Of neonates with cardiac murmurs, 75% had CHD.VSD is the commonest defect seen in 56.7% of cases.83 infants had CHD out of 11085 live births. Incidence of CHD in the study is 7.49/1000 live births.

Dr.M.Bansal et al[5]

Incidence of CHD is 6 per 1000 live births. Prevalence of heart murmur ranges from 0.6% to 47.4%. Prevalence of murmur is inversely

related to size of study. During routine post natal rounds 2603 healthy newborn were screened for murmur. Of 2603 neonates, 62 babies had murmur(2.3%). 28(45%) out of 62 babies had a cardiac malformation. Incidence of murmur was 23.81 per 1000 newborn. Incidence of CHD was 10.75 per 1000. If a murmur is detected there is 45% chance of there being significant cardiac malformation. Therefore when a murmur is detected in a neonate it necessitates prompt referral for echocardiography.

Laohprasitiporn D et al[6]

Total live births during the study period of one year were 11245. Murmur was detected in 83 cases in the first week of life. Incidence of heart murmur is 7.8/1000 live births. Out of 83 cases 49 had CHD. Incidence of CHD is 4.36/1000 live births.

Among innocent murmurs, echocardiogram showed no cardiac abnormality in 2 cases, mild tricuspid regurgitation in 2 cases, physiologic branch pulmonary stenosis in 4 cases and small size PDA in 26 cases.

Of babies with CHD incidence of VSD is 18.4%, PDA > 2mm is 16.3% and 1 baby had serious cardiac anomaly- Tetrology of Fallot.

Of neonates with CHD, 22(44.8%) were asymptomatic, 15(30.6%) were tachypneic, 8(16.4%) had cyanosis and 4(8.2%) had congestive

cardiac failure. Early diagnosis and proper management are needed to reduce morbidity and mortality in babies with CHD.

Z-D Du et al[7]

116 neonates with heart murmurs were evaluated with echocardiography. Age of these babies – 12 hours to 14 days. Of them 90 were term babies and 26 were preterm babies. 97(84%) neonates had CHD. 19(16%) had no cardiac anomaly. Out of 19, 7 had mild tricuspid regurgitation and 9 had physiological peripheral pulmonic stenosis. In 4 cases clinical diagnosis of VSD or pulmonic stenosis was changed to DORV, single ventricle, HLHS or TOF. Clinical evaluation could find out the presence or absence of heart disease to some extent. For lesion specific diagnosis echocardiography is necessary.

Nikyar B et al[8]

Prevalence rate of CHD is 8.6/1000 live births (9.96/1000male birth and 7.34/1000 female live birth). ASD is the commonest lesion-2.64/1000 LB. Combination of ASD and VSD – 1.28/1000 LB. PDA-1.28/1000 LB. Parents of 40 babies are of consanguineous marriage.

Kapoor R et al[9]

VSD – 21.3%

ASD – 18.9%

PDA – 14.6%

TOF - 4.6% (Commonest cyanotic congenital heart disease)

Maximum number of children with heart disease with (82.9%) were diagnosed between 0 and 3 years of age.

Frank JE et al[10]

The features which increase the liklihood of pathological murmur are diastolic murmur, grade 3 or higher murmur, harsh quality, associated with an abnormal S2 and when the maximal murmur intensity at upper left sternal border.

Amer Abdullah lardhi[11]

Incidence of CHD varies from 4/1000 to 50/1000 live births. 6333 neonates were studied. 87 neonates (1.37%) had murmur. 37 (42.5%) had a structural cardiac malformation. 24 had insignificant structural heart lesion (PFO, small PDA, mild peripheral pulmonary stenosis). 20 had structurally normal heart. The commonest defect is VSD (62%). VSD is seen in 23 babies, ASD- 5 babies, PDA – 3 babies, PS – 3 babies, PA -1

baby, AS- 1 baby, HOCM- 1 baby. 5.4% of babies required early cardiac intervention before they became symptomatic. Prevalence of cardiac murmur varies from 6/1000 to 770/1000. This wide range is due to difference in examiner's skill, experience, timing and frequency of examination.

Ainsworth et al[12]

7204 newborn babies were screened for murmur. All those with murmur were subjected to echocardiography. 46 neonates had cardiac murmur (6/1000). 25 neonates had cardiac malformation. Most common defect is VSD. If a murmur is heard there is 54% chance of there being cardiac disease. Prevalence of CHD in the study was 9 per 1000 live births. 6 in 1000 babies had murmur. Half of murmurs were due to structural heart disease. This examination led to recognition of 37% of all heart disease diagnosed in infancy.

Mohammed Monu Hossain et al[13]

Among 50 neonates with murmur 35 (68%) had structural heart defect. Among cases with structural heart defects 70.6% had significant heart defect and only 29.4% had physiological variant. Auscultation of heart in newborn period provides chance for early recognition of cardiovascular malformation.

Wren C. et al[14]

Of 1590 neonates with CHD 523 (33%) presented before physical examination because of symptoms due to non cardiac causes. On examining 1061 neonates abnormality is found in 476 (45%) babies. 170 of them were referred for diagnostic work up. 306 of discharged babies presented or died before 6 weeks. At 6 weeks on examining 252 of 569 babies abnormality found in 164 (65%) babies. Routine neonatal examination misses half of babies with CHD; examination at 6 weeks misses one third of cases.

Gregory J et al[15]

In their study one baby in 100 had murmur at 6 weeks. Nearly 50% of them were found to have structural heart disease. Hence referral of all babies with murmur at 6 weeks for echocardiography and expert opinion is recommended.

CONGENITAL HEART DISEASE

Definition:

Congenital heart diseases were defined by Mitchell et al as a structural abnormalities of heart or intrathoracic great vessels which is potentially of functional importance.

Incidence and prevalence of CHD

CHD occurs in 0.8% of live births i.e. 8 to 10 cases per 1000 live births.

In spontaneous abortuses	-	10-25%
In stillborns	-	3-4%
Premature infant	-	2% excluding PDA.

In preterm neonates (gestational age < 37 weeks) CHD is two to three times that found in term neonates.[16]

Diagnosis of CHD is established by 1 week of life in 40-50% of patients with CHD and by 1 month of age in 50-60% of cases.

With advances in medicine and surgical management, number of children with CHD surviving to adulthood has increased dramatically. In spite of these advancements in management, CHD is one of the major causes of death in children with congenital malformation. Timely

diagnosis and subsequent intervention particularly during newborn hospitalization are essential to reduce mortality associated with CHD.[17]

Frequency of major congenital heart lesions

Lesion	**% of all lesions**
Ventricular septal defect	25-30
Atrial septal defect	6-8
Patent ductus arteriosus	6-8
Coarctation of aorta	5-7
Tetrology of fallot	5-7
Pulmonary valve stenosis	5-7
Aortic valve stenosis	4-7
D-transposition of great vessel	3-5
Hypoplastic left ventricle	1-3
Hypoplastic right ventricle	1-3
Truncus arteriosus	1-2
Total anomalous pulmonary venous return	1-2
Tricuspid atresia	1-2

Single ventricle	1-2
Double outlet right ventricle	1-2
Others	5-10

(Excluding PDA in preterm neonates, bicuspid aortic valve, physiologic peripheral pulmonic stenosis and mitral valve prolapse).[1]

CLASSIFICATION OF CONGENITAL HEART DISEASE

ACYANOTIC CHD

i. Volume overload

- ✓ Left to right shunt
  - Atrial septal defect
  - Ventricular septal defect
  - AV septal defect
  - Patent ductus arteriosis
- ✓ Regurgitant lesion
  - Mitral regurgitation
  - Tricuspid regurgitation
  - Endocardial cushion defect
  - Aortic regurgitation
- ✓ Cardiomyopathy

ii. Pressure overload

- ✓ Ventricular outflow tract obstruction
  - Pulmonic stenosis
  - Aortic stenosis
  - Coarctation of aorta
- ✓ Ventricular inflow obstruction
  - Tricuspid stenosis
  - Mitral stenosis
  - Cor triatriatum
  - Obstruction to pulmonary venous return

CYANOTIC HEART DISEASE

- ✓ With decreased pulmonary blood flow
  - Tricuspid atresia
  - Tetrology of Fallot
  - Single ventricle with PS
- ✓ With increased pulmonary blood flow
  - Transposition of great vessels
  - Total anomalous pulmonary venous connection
  - Truncus arteriosus

DUCT DEPENDENT LESIONS

- Lesions which depend on duct to maintain *systemic circulation*
    - Coarctation of aorta
    - Critical aortic stenosis
    - Hypoplastic left heart syndrome
- Lesions which depend on duct to maintain *pulmonary circulation*
    - Pulmonary atresia
    - Critical pulmonary stenosis
    - Tricuspid atresia
    - Tetrology of Fallot
- Lesion which depends on duct for *mixing of systemic and pulmonary circulation*
    - Transposition of great vessels

Prostaglandin infusion is necessary to maintain ductus arteriosus patency and for survival of the patient. Closure of PDA can precipitate metabolic acidosis, seizures, cardiogenic shock, cardiac arrest and end organ damage.[21]

LESIONS PRESENTING AT DIFFERENT AGES[22]

Age on admission:*0-6 days* (n = 537)	Percentage of patients
D- Transposition of great arteries	19
Hypoplastic left ventricle	14
Tetrology of Fallot	8
Coarctation of aorta	7
Ventricular septal defect	3
Others	49

Age on admission*: 7-13 days*	Percentage of patients
Coarctation of aorta	16
Ventricular septal defect	14
Hypoplastic left ventricle	08
d-transposition of great vessels	07
Tetrology of fallot	07
Others	48

Age on admission: *14-28 days* (n=177)	Percentages of patients
Ventricular septal defect	16
Coarctation of aorta	12
Tetralogy of fallot	07
d-transposition of great vessels	07
Patent ductus arteriosus	05
Others	53

ETIOLOGY

Causes of most CHD are *unknown.* Most of the CHD were of *multifactorial* etiology –a combination of genetic predisposition and environmental stimuli.

Some of the CHD are related to chromosomal abnormality in particular Trisomies and Turner.

Syndrome	% of children with CHD
TRISOMY 18	90
Trisomy 21	50
Turner	40

Congenital infection

- Maternal rubella in first trimester is hazardous. It can predispose to PDA and pathological pulmonary artery stenosis.
- Cytomegalovirus, Herpes and Coxsackie virus B are suspected to be teratogenic. Infection by them may result in myocarditis in neonates.
- Maternal HIV infection may result in infantile cardiomyopathy.

Maternal medications and CHD

Amphetamine - VSD, PDA, ASD, TGA.

Phenytoin - PS, AS, COA, PDA.

Trimethadione - TGA, TOF, Hypoplastic left heart syndrome.

Lithium - Ebsteins anomaly.

Retinoic acid - Conotruncal anomalies.

Valproic acid- ASD, VSD AS, pulmonary atresia, COA.

Alcohol- VSD, PDA, ASD, and TOF.

Cigarette smoking - not proved to be teratogenic causes IUGR.

Maternal Conditions

1. Infants of Diabetic Mothers have higher incidence of structural heart defect- TGA, VSD, PDA.

2. SLE in mother is associated with higher incidence of congenital heart block in newborn.

3. Incidence of CHD is 1% in general population but it is 15% if the mother has CHD.

Birth history

-Perinatal complications like to toxemia, asphyxia and fetal distress can lead to generalized cardiomyopathy.

-It important to note Apgar score, cyanosis and perfusion status.[18]

Birth weight

-SGA babies suggest intrauterine infections or use of drugs.

-LGA babies show a higher incidence of cardiac anomalies. Infants with TGA have a birth weight higher than average.

Family history

- History of congenital heart disease or cardiomyopathy in mother, father and siblings is noted.

-There is an overall increased risk for CHD when a first degree relative has CHD.[19, 20.]

Environmental factor

- There is a higher incidence of PDA and ASD in children born at high altitude.

HISTORY

After getting a thorough antenatal and natal history, history of babies breathing and feeding pattern should be taken. History of rapid breathing, cyanosis, chest retraction, volume taken per feeding, suck-rest-suck cycle, diaphoretic while sucking should be noted.

PHYSICAL EXAMINATION

On general examination of the baby extra cardiac anomalies should be noted. Following are some of the heart defects commonly associated with extra cardiac malformations.

Extra Cardiac Anomalies[23]	**Likely Congenital Cardiac Lesion**
Absence of radius or ulna	VSD
Syndactyly , polydactyly	VSD
Trisomy 21	ASD of endocardial cushion type, VSD
Arachnodactyly	ASD
Turner syndrome	COA, PS, AS
Ellis-van Crevald syndrome	ASD, single atrium
Rubella syndrome	PDA, Pulmonic stenosis
Moon facies and hypertelorism	Pulmonic stenosis.
Holt Oram syndrome	Familial ASD
Marfan syndrome	Aortic or pulmonary artery dilation
Hurler syndrome	MR or AR
Trisomy 13-15	VSD
Trisomy 17-18	VSD, PDA

Respiratory distress

- Neonates comfort level should be observed.
- Tachypnea may suggest shunt lesions and Cyanotic heart disease.
- Grunting and dyspnea often accompany left sided obstructive lesion.

Colour of the child

- True cyanosis requires desaturation of 5g% of hemoglobin.
- It is difficult to detect unless the saturation is 85% or lower
- Clinically it is best observed in tongue.
- Clinical assessment of cyanosis is inadequate and pulse oximetry should always be performed.
- When 0_2 saturation in lower limb is lower compared to right upper limb, right to left shunting across ductus arteriosus should be suspected.
- It may be associated with persistant pulmonary hypertension or COA.

Heart rate

In neonates with heart rate lower than 90 beats per minute or more than 160 beats per minute ECG is performed to rule out arrhythmia.

- sinus tachycardia is seen in large left to right shunts and myocarditis.

- SVT may be seen in Ebsteins anomaly.

- Bradycardia is associated with long QT syndrome as well as congenital AV block.[24, 25, 26, 27.]

Pulse

- Rate , regularity , quality of pulsations should be checked.
- Upper and lower limb pulses are simultaneously checked and any delay is noted.
- Weak leg pulses and strong arm pulses suggest COA.
- Bounding pulses suggest PDA , AR or persistant truncus arteriosus
- Weak, thready pulse suggest cardiac failure or circulatory shock
- Cardiogenic shock must be differentiated from other causes of shock like sepsis.
- In newborn with shock, cardiomegaly indicates cardiac etiology.[28]

Blood pressure

- BP is not routinely measured in healthy new born

- It should be measured when there is suspicion of COA

- systolic pressure difference of 20mm Hg more in upper limb than lower limb is a clue to the diagnosis of COA.[29]

CVS EXAMINATION

INSPECTION

- Chest deformities should be noted.
- A hyperdynamic precordium suggest volume overload.
- A precordial bulge denotes cardiac enlargement.
- Apical impulse position is noted.

PALPATION

Apical impulse

- Its location and diffuseness should be noted
- In newborn, it is at 4th intercostal space just to the left of mid clavicular line
- When it is displaced laterally or downward suggests cardiac enlargement.

Thrill

- Thrills are vibratory sensations.
- These represent palpable manifestation of loud murmur.

- When murmur is associated with thrill, it points towards structural heart disease.

AUSCULTATION

- It is the most cost effective tool for the cardiac diagnosis

- A binaural stethescope is used with a bell and a diaphragm

- Bell picks up low frequency events

- Diaphragm is selectively better suited for detecting high frequency events

Heart sounds

Should be auscultated and analysed before the analysis of heart murmurs.

First heart sound

- Associated with closure of mitral & tricuspid valve.
- Best heard at apex or lower left sternal border.
- Splitting of s_1 is infrequent.

Second heard sound

- It is heard in upper left sternal border

- S_2 splitting and intensity of pulmonary closure component (P_2) should be noted

- Abnormal splitting may be in the form of wide splitting, narrow splitting, a single s_2 or paradoxical splitting of s_2

- Usually A_2 is louder than P2. When P2 is of increased intensity it suggests pulmonary hypertension, when p_2 is of decreased intensity it may be due to severe PS, TOF of TA.

Third heart sound

- It is a low frequency sound related to rapid filling of the ventricle
- Best heard at apex or lower left sternal border
- Commonly heard in normal children and young adults.
- Loud S_3 is abnormal and heard when there is reduced ventricular compliance.

Fourth Heart Sound

- It is a low frequency sound.
- When present it is always pathologic
- Heard in cases of CCF or when there is reduced ventricular compliance.

Systolic and Diastolic Sounds

Ejection click

- It occurs at the time of ventricular ejection onset.
- Follows S_1 closely.
- Heard at the base (either side of upper sternal border).
- Associated with stenosis of semi lunar valves.

Mid systolic click

- Heard in cases of mitral valve prolapse at the apex.

Diastolic opening snap

- Originates from a stenosis of AV valve.
- Heard at the apex or lower left sternal border.

HEART MURMURS

Heart murmurs are abnormal sound heard due to turbulent blood flow. These are blowing, whooshing or rasping sounds heard between the heart sounds.

Heart murmur should be described in terms of intensity, timing, location, transmission, and quality.

Heart murmurs are a common finding in infants and arise from normal flow patterns with no structural abnormalities of heart; conversely murmur may be created by abnormal flow patterns in heart and vessels resulting from congenital heart abnormalities. If a murmur is heard there is a 54% chance of there being a cardiac malformation. So careful evaluation of murmur should be done.[30, 31.]

Intensity of murmur

It is graded from 1 to 6 based on recommendation of Samuel A Levine in 1933.

Grade 1 – Barely Audible

Grade 2 – Soft but easily audible (medium intensity)

Grade 3 – Moderately loud but not accompanied by a thrill.

Grade 4 – Louder and associated with a thrill

Grade 5 – Audible with the stethoscope barely on the chest.

Grade 6 – Audible with the stethoscope off the chest.

Classification of heart murmur

Based on timing of heart murmur with respect to S_1 and S_2 heart murmur is classified into systolic, diastolic, and continuous murmur.

SYSTOLIC MURMUR

Most of the cardiac murmurs are systolic in nature. It is classified as ejection and regurgitant murmur.

Ejection systolic murmur

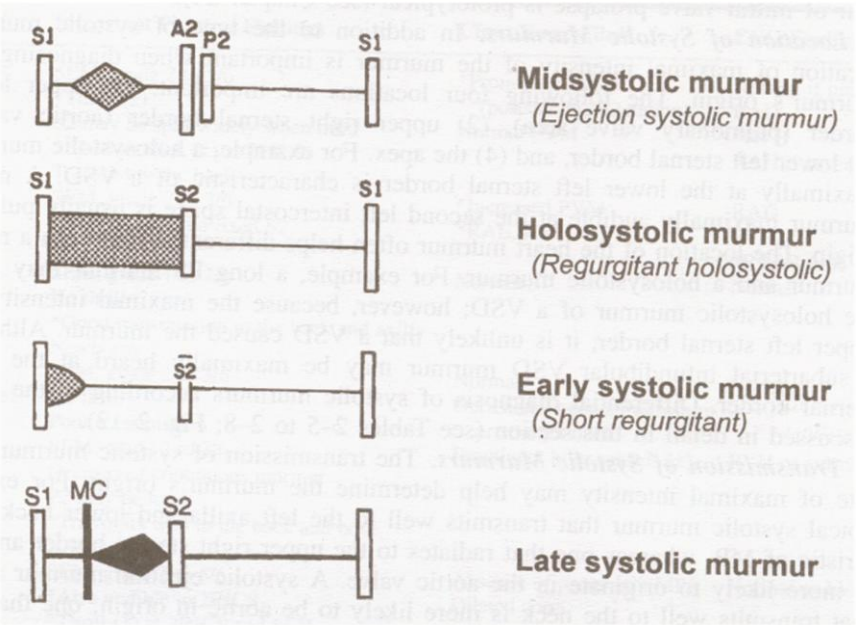
Also known as stenotic or crescendo – decrescendo murmurs. There is a gap between S_1 and onset of murmur. These occur due to blood flow through stenotic or deformed semi lunar valve or due to increased flow through normal semi lunar valve. So they are audible at 2^{nd} left or 2^{nd} right intercostal space.

Regurgitant systolic murmur

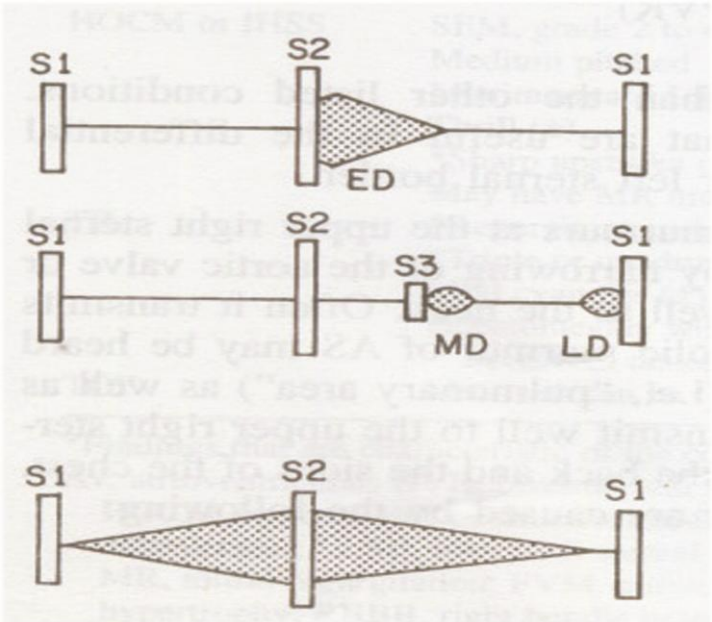
There is no interval between S_1 and the onset of regurgitant murmur. It lasts throughout the systole. Hence it is named pansystolic or holosystolic murmur.

These are due to flow of blood from a chamber which has got more pressure throughout the systole than the receiving chamber. Three conditions which cause holo systolic murmur are VSD, MR and TR.

SYSTOLIC MURMUR



DIASTOLIC MURMUR



Early diastolic murmur

Mid-diastolic murmur

Continuous murmur

DIASTOLIC MURMUR

Occur between S_2 and S_1 in relation to heart sounds they are classified into early diastolic, mid diastolic and pre systolic.

Early Diastolic Murmur

These decrescendo murmurs occur early in diastole, immediately after S_2 caused due to incompetence of aortic or pulmonary valve.

Mid diastolic murmur

They start with loud S_3 and are always low pitched. These murmur are caused by mitral or tricuspid valvular stenosis and best heard at the apex. MDM are associated with MS or a large left to right shunt (VSD or PDA). In VSD or PDA, MDM occurs due to relative mitral stenosis secondary to large flow across the valve.

Pre systolic murmur

It is caused by flow through atrioventricular valves during ventricular diastole. Active atrial contraction that ejects blood into ventricle through narrow valve results in presystolic or late diastolic murmur.

CONTINUOUS MURMUR

These start in systole and continue through the S_2 into all or part of diastole. These are caused by :

1. Aorto pulmonary or arteriovenous connection – PDA, AV fistula.
2. Disturbance of flow patterns in veins (e.g. Venous hum).
3. Disturbance of flow patterns in arteries (e.g. COA, PA stenosis).

INNOCENT HEART MURMURS

These are also known as *functional murmurs*. These arise from cardiovascular structure in the absence of anatomic abnormalities. Innocent murmurs are common in infants and children.

Characteristics of innocent murmur

- no thrill

- grade 1-2/6

- normal femoral pulses

- no respiratory distress

- no cyanosis

- no hepatomegaly

- not associated with dysmorphic features or other congenital anomaly

- mostly associated with normal ECG and X-ray findings.

Innocent murmurs are classified into:

i. **Classic vibratory murmur**

- Described by Still in 1909.
- Common in the age group 3-6 yr.
- It is mid systolic grade 2 to 3/6, low frequency and of musical quality.
- It is thought to be generated by vibration of normal pulmonary leaflets at their attachments.
- Usually confused with murmur of VSD.

ii. **Pulmonary flow murmur of childhood**

- Common in age group 8-14 yr.
- It is mid systolic grade 1 to 3/6.
- It is due to exaggeration of normal ejection vibration within the pulmonary trunk.
- This murmur is exaggerated in pectus excavatum and kyphoscoliosis.

- This may be confused with murmur of ASD or pulmonary valve stenosis.

iii. <u>Pulmonary flow murmur of newborns</u>

- Commonly occurs in newborn and more often in low birth weight babies.
- Disappears by 3 to 6 months of age.
- Grade 1 to 2/6 at the upper left sternal border.
- In the fetus, the branches of pulmonary artery are relatively hypoplastic.
- So when increased flow occurs through these vessels in newborn turbulence is transmitted along branches of PA.
- It is confused with murmur of organic PA stenosis.

iv. <u>Venous hum</u>

- Common in age group 3-6 Yrs.
- It is a continuous murmur due to turbulence in jugular venous system.
- Well heard at the right or left infraclavicular and supraclavicular areas.
- Confused with murmur of PDA.

v. Carotid bruit

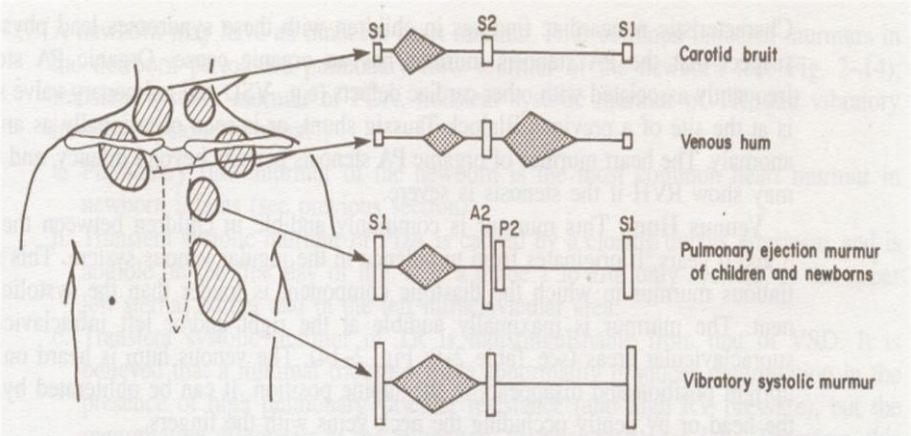
(supraclavicular systolic murmur)

- It is an early systolic ejection murmur heard over carotid arteries.
- It originates from turbulence in brachiocephalic or carotid arteries.
- Grade 2 to 3/6, associated with faint thrill
- May be found in children of any age.
- Murmur of AS is transmitted to carotid arteries and so it must be differentiated from carotid bruit.[32]

IMPORTANCE OF FOLLOW UP OF BABIES AT 6 WEEKS OF LIFE

Baby may have insignificant lesion at birth causing murmur. During their follow up for vaccination at 6 weeks they should be auscultated for murmur. Most of the innocent murmurs disappear. Organic murmurs persist. Echocardiogram should be done for all babies who had positive echo finding at birth and who have murmur at 6 weeks. Presence of heart disease needs proper management and referral. Absence of heart disease allays the unnecessary fear of parents. When a murmur is detected at 6 weeks of life the likelihood of structural heart disease is high.

INNOCENT MURMURS



SPECIAL FEATURES OF CARDIAC EXAMINTATION OF NEWBORNS

The difference is caused due to normal RV dominance and elevated pulmonary vascular resistance in early neonatal period.

Normal findings in a neonate

1. Heart rate is faster in newborns
2. RV is hyperactive so the point of maximum impulse is at lower left sternal border rather than at apex.
3. S_2 may be single.
4. 4 common innocent murmur heard in newborn are
    - Pulmonary flow murmur of the newborn.
    - Transient systolic murmur of PDA.
    - Transient systolic murmur of TR.
    - Vibratory innocent systolic murmur.

Abnormal physical findings in a neonate

1. Cyanosis not improving with oxygen suggests a cardiac abnormality.
2. Decreased peripheral pulses in lower limbs are suggestive of COA. Weak peripheral pulses throughout indicate HLHS or circulatory shock. Bounding peripheral pulses is found in PDA or persistent truncus arteriosus.
3. Tachypnea (Rate > 60/ min) with or without retraction suggest a cardiac abnormality.
4. Hepatomegaly suggest a heart disease. Midline liver indicate asplenia or polysplenia syndrome.
5. Irregular rhythm and abnormal heart rate are suggestive of cardiac abnormality.
6. Presence of heart murmur could be a sign of CHD. Most pathologic murmurs are heard during first month of life except ASD.
    i. Murmur of stenotic lesions (AS, PS) and AVvalve regurgitation are heard immediately after birth. These murmurs are not affected by the level of pulmonary vascular resistance.

ii. Murmur of VSD become audible by 1 to 2 weeks of life when pulmonary vascular resistance falls and shunt occurs.

iii. Murmur of ASD is audible by 1 yr of life when the compliance of RV improves to allow significant atrial shunt.

7. Even in absence of heart murmur a neonate may have hazardous heart defect that needs immediate attention and intervention (TGA, pulmonary atresia). Murmur may not be heard in CCF due to myocardial dysfunction.

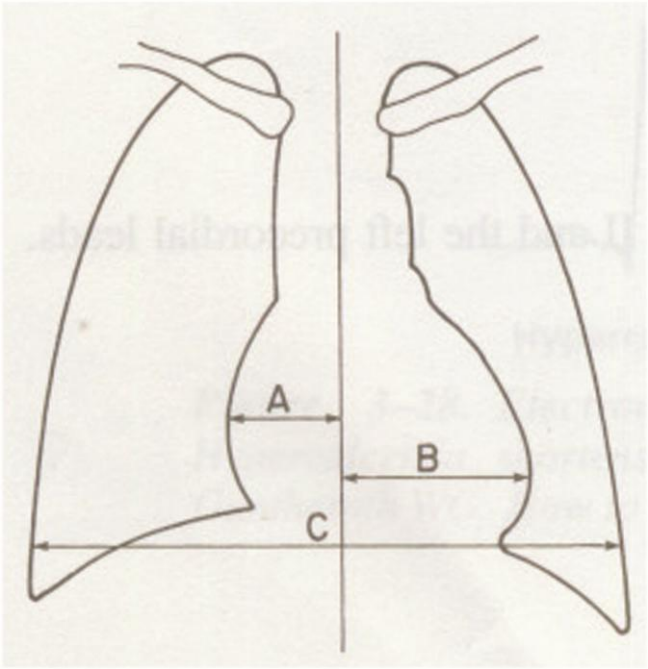
X-RAY CHEST

It is an essential part of cardiac evaluation. It furnishes the following details

- Heart size and silhouette
- Enlargement of specific cardiac chamber
- Pulmonary blood flow ,pulmonary vascular marking s
- Lung parenchyma
- Spine, bony thorax
- Abdominal situs

Heart size

Cardio thoracic ratio is the simple way to determine heart size in children.



CT ratio = (A+B)/C

A&B –Maximal Cardiac Dimension to the right and left of midline.

C-widest internal diameter of the chest.

Cardiomegaly is considered when CT ratio is more than 0.5. But it is not applicable to new born in whom a good inspiratory film cannot be obtained.

Normal cardiac silhouette.

- Right cardiac silhouette is formed by SVC and inferiorly by right atrium.
- Left cardiac border is formed by aortic knob,main pulmonary artery and left ventricle.

In new born, a typical normal cardiac silhouette is rarely seen due to presence of a large thymus and films are obtained during expiration.

Abnormal cardiac silhouette

1. Boot shaped heart with decreased pulmonary blood flow is seen in TOF and tricuspid atresia.
2. Egg-shaped heart-is seen TGA. heart silhouette is narrow-waisted due to absence of thymus and abnormal relationship between great arteries.
3. Snowman sign-with increased pulmonary flow is seen in supracardiac type of TAPVR.

Individual chamber enlargement

Left atrial enlargement

Mild enlargement is seen In Lateral View. LA enlargement produce double density on PA view. Left atrial appendage become prominent on left cardiac border. Left main stem bronchus is elevated. Indentation of esophagus could be seen in barium swallow film.

Left ventricular enlargement

In PA view, apex is shifted outward and downward.

Right atrial enlargement

This is seen as an increased prominence of lower right cardiac silhouette

Right ventricular enlargement

In may not be seen in PA view, best recognized in lateral view as filling of retrosternal space.

Size of great arteries

Prominent main pulmonary artery segment

Seen in post stenotic dilation, increased flow through PA and increased pressure in PA (e.g.pulmonary hypertension)

Hypoplasia of pulmonary artery

Concave main PA segment is seen in TOF and tricuspid Atresia

Dilation of aorta

Seen in TOF, AS and PDA,COA,Marfan Syndrome.

Pulmonary vascular markings

Increased pulmonary blood flow

- Right and left PA appear enlarged
- Vascularity extend into lateral third of lung field and to lung apices
- In acyanotic child , it indicates ASD,VSD,PDA or PAPVR.
- In Cynatic Infant, It indicates TGA ,TAPVR,HLHS,persistant truncus arteriosus or a single ventricle.

Decreased pulmonary blood flow

- Seen in TOF ,pulmonary atresia and tricuspid atresia.
- Hilum is small, lung fields are black and vessels are thin.

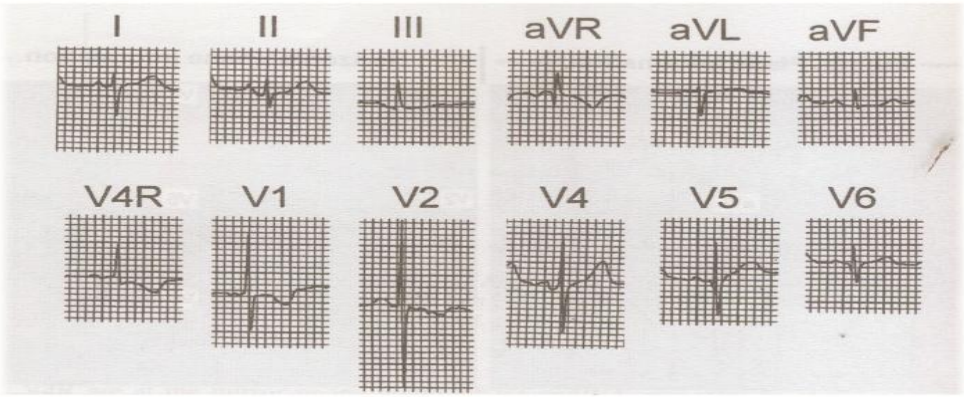
Pulmonary venous congestion

It is characterized by indistinct margin of pulmonary vasculature. It is secondary to left ventricular failure or obstruction to pulmonary venous drainage. kerly B lines are caused by engorgement and dilation of lymphatics and edema of interlobular septae.[32]

ELECTRO CARDIOGRAPHY

Hypertrophies of chamber and ventricular conduction disturbances are common forms of ECG abnormalities in CHD.

ECG of a neonate is quite different from that of normal adult. RV dominance is seen in neonates. It gradually changes to LV dominance as the baby grows. By 3 years of age, childs ECG resemble that of adults.



ECG of normal 1 week –old infant.

RV dominance is seen in ECG as right axis deviation and large anterior QRS forces and deep S waves in lead I and left precordial leads (V5+V6)

T Waves in V1 is usually negative.

Abnormal ECG patterns such as AV and intraventricular conduction disturbance, ventricular hypertrophy signs points towards presence of heart disease.[33]

ASD

ECG shows right axis deviation and right ventricular hypertrophy in ostium secundum ASD. The characteristic configuration of precordial lead V_1 is $rsR^{'}$ seen in almost 90% of patients. left axis deviation of more than 30^{o} is suggestive of ostium primum ASD.

VSD

When VSD is small, ECG is normal. when VSD is moderate, LVH and occasional LAH is seen. When VSD is large, biventricular hypertrophy is seen. In cases with pulmonary vascular obstructive disease RVH is seen.

PDA

LVH is seen in small to moderate PDA. biventricular hypertrophy is seen with large PDA. If there is development of pulmonary vascular obstructive diseases, RVH is seen.

Endo cardial cushion defect

The characteristics ECG tracing of this defect shows superior QRS axis with QRS axis between -40 and -150 degrees .PR interval is prolonged. RVH or RBBB is present.

Pulmonary stenosis

In moderate PS, right axis deviation and right ventricular hypertrophy is seen. Neonates with critical PS may show LVH. This is due to hypoplastic RV and large left ventricle.

Aortic stenosis

LVH may be present.

Coarctation of aorta

In infants, RVH or RBB are present, LVH is seen in order children.

D-TGA

There is rightward QRS axis (+90 to +200 degree). RVH is seen. after 3 days of life an upright T wave in V_1 may suggest RVH.

L-TGA

The characteristics features is absence of Q waves in V_5 and V_6 or the presence of Q waves in V_4 or V_1. This happens because the direction of ventricular septal depolarization is from the embryonic LV to RV. AV block of varying degree are seen.

TOF

Right axis deviation and RVH is usually present. In acyanotic form, QRS axis is normal and BVH is seen.

TAPVC

RVH and occasional RAH are present.

Tricuspid atresia

Superior QRS axis (between 0 and -90 degrees) is characteristic.

Pulmonary Artesia

QRS axis is normal LVH is seen RVH is seen in infants with a relatively large RV cavity.

Hypo plastic left heart syndrome

ECG shows RVH large R waves may be seen in V_5 and V_6 because these leads record over dilated RV and not over hypo plastic LV.

Ehstein's Anomaly

RBBB and RAH are characteristics findings. first degree AV block is seen in 40% of patients.

Persistant trunsus arteriosis

QRS axis is normal (+50 to +120degrees) .BVH is seen.

Persistent pulmonary hypertension of the newborn.

Usually normal ECG is seen for age. occasional RVH may be present.

From the literature it was found that diagnosis based on ECG and X Ray were 42% correct. It was most accurate with diagnosis of TGA, TOF, Coarctation of aorta and VSD.[34.]

ECHOCARDIOGRAPHY

Echocardiography is a safe and non-invasive test and it is very helpful in the diagnosis and management of heart disease.

The utility of echo has improved with the incorporation of pulsed, continuous and color Doppler flow studies. The combination gives precise anatomic and functional information for most types of congenital heart defect.[35]

Uses of Echocardiography

- To evaluate structural defect in congenital heart disease
- Estimate intra-cardiac pressure and gradient across stenotic valves and vessels.
- Quantitate systolic and diastolic function of chambers
- Determine the direction of flow in a shunt.
- Examine the integrity of coronary arteries.
- Detect the vegetations of infective endocarditis.
- Detect the presence of pericardial fluid, cardiac tumours and chamber thrombi.
- Assist in performing interventional procedures.
- Transesophageal echocardiography is used to monitor ventricular function during surgical procedure and can assess the results of surgical repair of CHD.

M – MODE ECHOCARDIOGRAPHY

It shows a one – dimensional slice of cardiac structure varying over time. It measure cardiac wall thickness, chamber size fractional shortening and wall thickening. It asses the motion of intra cardiac structure such as valves, the walls and septa. Some of the indices calculated from echo are

1) $FS\ (\%) = \frac{LVED - LVES}{LVED}$

FS – Fractional shortening

LVED – Left Ventricular dimension at End Diastole

LVES – Left Ventricular dimension at End Systole

2) $EF\ (\%) = (LVED)^3 - (LVES)^3 / (LVED)^3 \times 100$

EF – Ejection Fraction

LVED – Left ventricular dimension at End Diastole

LVES – Left Ventricular dimension at End Systole

More useful indices of cardiac function can be obtained with the assistance of echocardiography.

2 – DIMENSIONAL ECHOCARDIOGRAPHY

The contracting heart is imaged in real time with 2-D echocardiography. It is obtained from four transducer location - parasternal, apical, subcostal and suprasternal notch position.

2-D echo has replaced cardiac angiography for pre – operative diagnosis of most of the CHD.

For parasternal view, the transducer is applied to the left parasternal border in the second, third or fourth space with the patient in left lateral decubitus position.

Transducer is placed over cardiac apex when patient is in left lateral decubitus position for apical view.

APICAL FOUR –CHAMBER VIEWS

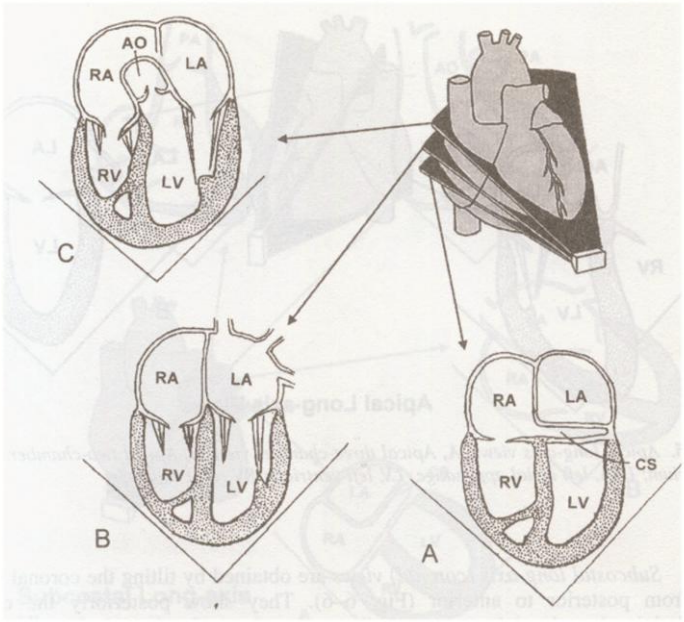


Diagram of two –dimensional echo views obtained with the tranducer at the apical position, A, A posterior plane view showing the coronary sinus, B, The standard apical four-chamber view.C, the apical five –chamber view is obtained with further anterior angulation of the transducer, AO, aorta; CS, coronary sinus; LA left atrium; LV, left ventricle; RA, right atrium; RV right ventricle.

For subcostal view, transducer is positioned from subxiphoid area with the patient in supine position.

For suprasternal view, transducer is applied to the suprasternal notch. It helps in the evaluation of anomalies in ascending and descending aorta, aortic arch, size of pulmonary arteries, anomalies of systemic veins and pulmonary veins.

DOPPLER ECHOCARDIOGRAPHY

It gives us the picture of blood flow profiles. It is based on the change in frequency imparted to a sound wave by the movement of RBCs.

The two commonly used Doppler techniques are continuous wave and pulsed wave. The clinical application expands when these two techniques are combined.

Doppler echo detects the presence and direction of cardiac shunts; indentifies stenosis on regurgitation of blood vessels; helps in determining pressure in various compartments; estimate diastolic function of ventricles.

The assessment of a baby for the presence of heart diseases can be done with nadas criteria.

NADAS CRITERIA

The criteria are divided into major and minor criteria. Presence of one major or two minor are required to establish presence of cardiac diseases.

MAJOR CRITERIA

1. Systolic murmur grade III or more in intensity.
2. Diastolic murmur
3. Cyanosis
4. Congestive cardiac failure.

MINOR CRITERIA

1. Systolic murmur less than grade III in intensity.
2. Abnormal second heart sound.
3. Abnormal electrocardiogram.
4. Abnormal X-ray chest showing cardiomegaly in a good inspiratory film.
5. Abnormal blood pressure.

MATERIALS AND METHODS

Study place

Govt. Mohan Kumaramangalam Medical College Hospital, Salem-636001.

Study period

One year (October 2011 to September 2012)

Study design

Hospital based prospective study

Study population

All new born babies born in GMKMCH during the study period.

Inclusion criteria

- All term babies delivered in our hospital with cardiac murmurs on clinical examination were included into the study**.**

Exclusion criteria

- Preterm babies.
- All sick and moribund babies who cannot be completely evaluated

Study protocol:

Ethical committee clearance was obtained to conduct the study in our hospital. Informed consent was obtained from parents or caregivers before including them in the study. There is no added risk or harm to the baby because of the study. A data collection sheet (proforma) was filled for each neonate.

A detailed history was elicited for all recruited babies with heart murmur and was thoroughly examined for clinical signs. It was followed later by ECG and Chest X-ray. Following which all neonates underwent an echocardiographic examination by Cardiologists. Then neonates were classified as having innocent murmur and structural heart defect. The Latter were further subdivided into physiological variant or with significant heart disease. Babies with any abnormality were followed up at 6 weeks of age for persistence of murmur and heart disease.

Statistical analysis:

All statistical analysis were done using ***SPSS 20 version*** (statistical package for social sciences)

Appropriate test of significance (chi square test) used wherever necessary for testing correlation between different variables.

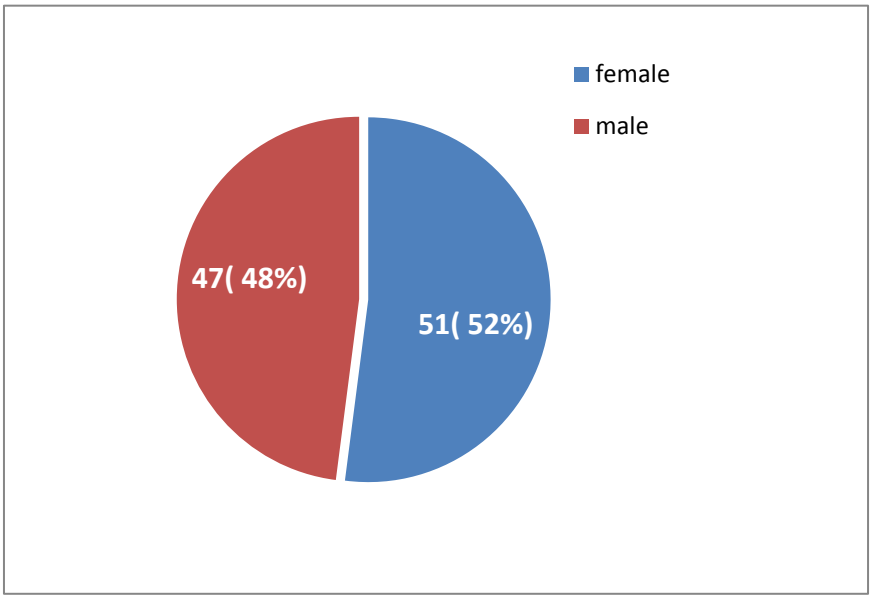
A P value of 0.05 or less was considered for statistical significance.

OBSERVATION AND RESULTS

Total number of live births during the study period	4116
No. of neonates with murmur	98
No. of neonates with structural heart disease	52
No. of neonates with heart disease after 6 weeks follow up	27

Incidence of murmur in first week of life	23 / 1000 live births
Incidence of congenital heart disease at birth	12.6 /1000 live births
Percentage of structural heart disease in neonates with murmur	53.06 %
Incidence of congenital heart disease at 6 weeks	6.6 /1000 live births

Graph 1: Gender distribution



Among 98 neonates 47 were males and 51 were females.

Graph 2: Birth weight distribution

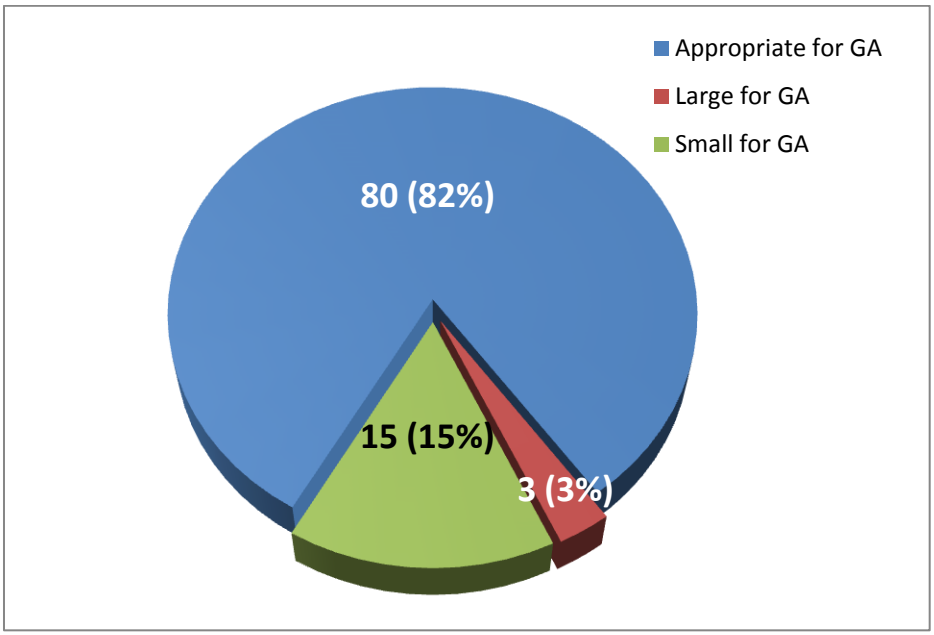


Table 1: Correlation between gender distribution and CHD at birth and at 6 weeks

Sex	CHD at birth n (%)		HD at 6weeks n (%)		
	Normal	Congenital Heart disease	Not Applicable	NO	YES
Female	24 (52.2%)	27 (51.9%)	25 (47.2%)	8 (44.4%)	18 (66.7%)
male	22 (47.8%)	25 (48.1%)	28 (52.8%)	10 (55.6%)	9 (33.3%)

Sex vs. CHD at *birth* –chi square p value 0.98 (not significant)
Odds ratio for female – 1.010
95% confidence interval- 0.457 to 2.235 (not significant)

Sex vs. CHD at *6 weeks* –chi square p value 0.139 (not significant)
Odds ratio for female in comparison to male – 2.50
95% confidence interval- 0.733 to 8.524 (not significant)

Correlation between sex distribution and CHD at birth and 6 weeks is not significant.

Table 2: Correlation between birth weight and CHD at birth and at 6 weeks

weight	CHD at birth		HD at 6weeks		
	Normal	Congenital Heart disease	Not Applicable	NO	YES
AGA	36(78.3%)	44 (84.6%)	42 (79.2%)	17 (94.4%)	21(77.8%)
LGA	1 (2.2%)	2 (3.8%)	1 (1.9%)	0 (0.0%)	2 (7.4%)
SGA	9 (19.6%)	6 (11.5%)	10 (18.9%)	1 (5.6%)	4 (14.8%)
Total	46(100%)	52(100%)	53(100%)	18(100%)	27(100%)

Weight vs. CHD at birth –chi square p value 0.504 (not significant)

Reference category –AGA

Odds ratio for LGA – 1.636

P value -0.692 (not significant)

95% confidence interval- 0.143 to 18.784 (not significant)

Odds ratio for SGA – 0.545

P value -0.290 (not significant)

95% confidence interval- 0.177 to 1.677 (not significant)

Weight vs CHD at 6 weeks –chi square p value 0.283 (not significant)

Odds ratio for Small or large weight for GA- 4.857

(in comparison to AGA)

95% confidence interval- 0.532 to 44.341 (not significant)

Correlation between birth weight and CHD at birth and at 6 weeks is not significant.

Table 3: Maternal risk factors

Maternal risk factor	Frequency	Percent
Nil	72	73.5
Diabetes	3	3.1
DM + Polyhydramnios	2	2.0
Polyhydramnios	11	11.2
Oligohydramnios	8	8.2
Intra-uterine infection	2	2.0
Total	98	100.0

Graph 3 : Maternal risk factors

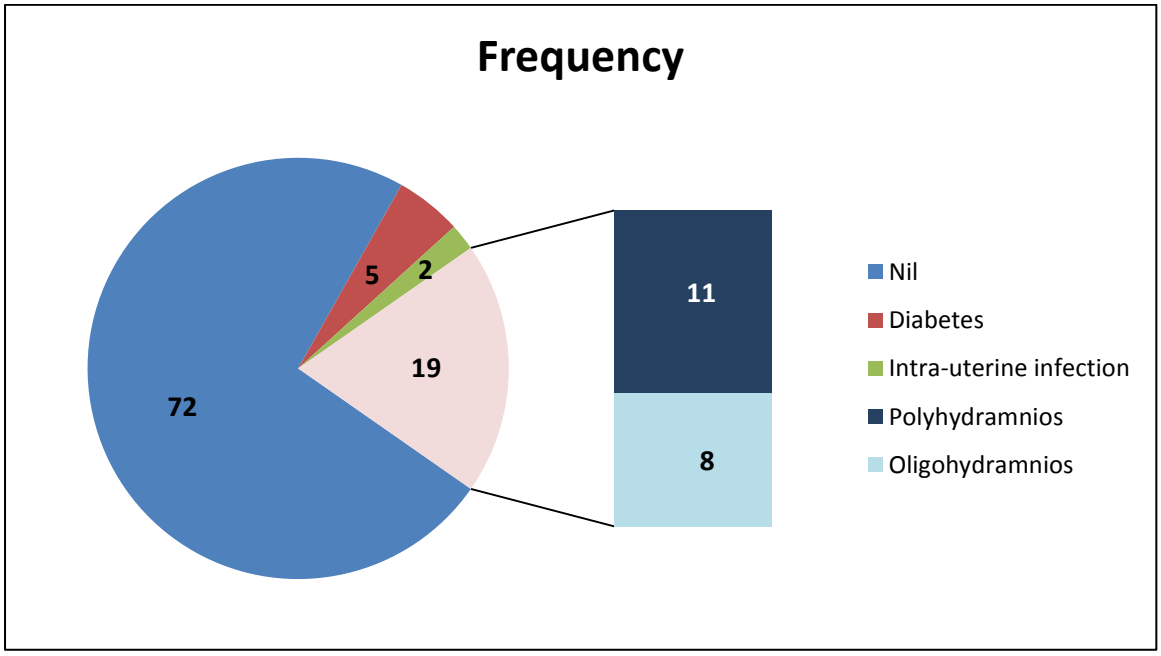


Table 3A: Correlation between maternal risk factor and CHD at birth and at 6 weeks

Maternal risk factor	Echo at birth		Echo at 6weeks		
	Normal	Congenital Heart disease	Not Applicable	NO	YES
Nil	37 (80.4%)	35 (67.3%)	42 (79.2%)	13 (72.2%)	17 (63.0%)
Diabetes	2 (4.3%)	1 (1.9%)	2 (3.8%)	0 (0.0%)	1 (3.7%)
DM + P.h	0 (0.0%)	2 (3.8%)	0 (0.0%)	0 (0.0%)	2 (7.4%)
P.h	3 (6.5%)	8 (15.4%)	3 (5.7%)	4 (22.2%)	4 (14.8%)
O.h	4 (8.7%)	4 (7.7%)	6 (11.3%)	1 (5.6%)	1 (3.7%)
IUI	0 (0.0%)	2 (3.8%)	0 (0.0%)	0 (0.0%)	2 (7.4%)
Total	46 (100.0%)	52 (100.0%)	53 (100.0%)	18 (100.0%)	27 (100.0%)

(P.h - Polyhydramnios)
(O.h - Oligohydramnios)
(IUI - Intra-uterine infection)

Risk factors vs CHD at birth –chi square p value 0.276 (not significant)
Odds ratio for infants with any risk factor- 1.997
(in comparison to no risk factor)
95% confidence interval- 0.787 to 5.065 (not significant)

Riskfactors vs CHD at 6 weeks –chi square p value 0.566 (not significant)
Odds ratio for infants with any risk factor- 1.529
(in comparison to no risk factor)
95% confidence interval- 0.419 to 5.577 (not significant)

Correlation between maternal risk factor and CHD at birth and at 6 weeks is not significant.

Table 4: Positive family history

family history	Frequency	Percent
No Family history	67	68.4
Consanguinity	20	20.4
consanguinity & CHD in mother	1	1.0
consanguinity+CHD in sibling	2	2.0
CHD in mother	5	5.1
CHD in father	1	1.0
CHD in sibling	2	2.0
Total	98	100.0

Graph 4: Family history distribution

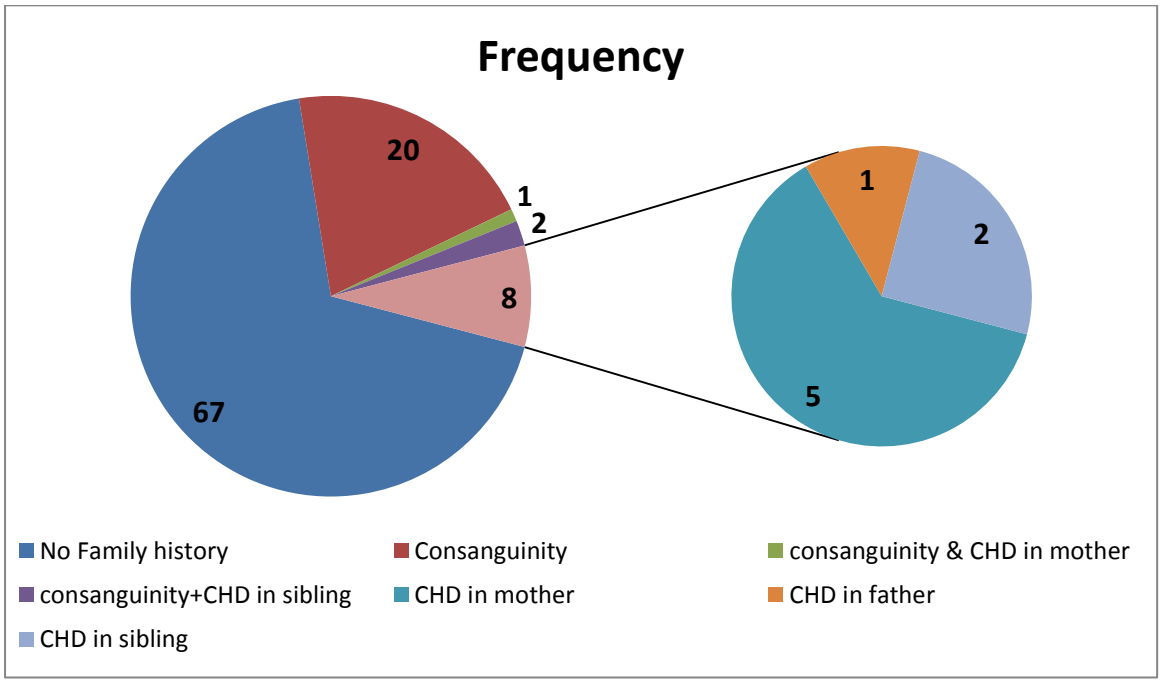


Table 4A: Correlation between Family risk factors and CHD at initial evaluation and CHD at 6 weeks

Family history	Echo at 1st week		CHD at 6weeks		
	Normal	Congenital Heart disease	Not Applicable	NO	YES
No Family history	37 (80.4%)	30 (57.7%)	39 (73.6%)	14 (77.8%)	14 (51.9%)
Consanguinity	6 (13.0%)	14 (26.9%)	10 (18.9%)	3 (16.7%)	7 (25.9%)
consanguinity & CHD in mother	0 (0.0%)	1 (1.9%)	0 (0.0%)	1 (5.6%)	0 (0.0%)
consanguinity + CHD in sibling	1 (2.2%)	1 (1.9%)	2 (3.8%)	0 (0.0%)	0 (0.0%)
CHD in mother	1 (2.2%)	4 (7.7%)	1 (1.9%)	0 (0.0%)	4 (14.8%)
CHD in father	0 (0.0%)	1 (1.9%)	0 (0.0%)	0 (0.0%)	1 (3.7%)
CHD in sibling	1 (2.2%)	1 (1.9%)	1 (1.9%)	0 (0.0%)	1 (3.7%)
Total	46	52	53	18	27

Family history vs CHD at birth –chi square p value 0.016 (significant)

Odds ratio for infants with any Family history - 3.015 P VALUE -0.018 (in comparison to no Family history)

95% confidence interval- 1.210 to 7.511 (significant)

Family history vs CHD at 6 weeks –chi square p value 0.079 (not significant)

Odds ratio for infants with any Family history - 3.250

(in comparison to no Family history)

95% confidence interval- 0.848 to 12.454 (not significant)

Correlation between positive family history and CHD *at birth is significant.* Correlation between positive family history and CHD *6 weeks is not significant.*

Table 5: Symptoms observed in study population

Symptom	Number
Cyanosis	6
Fast breathing	16
Feeding difficulty	9
Sweating	3

Table 5A :Correlation between presence of symptom and CHD at birth

Presence of symptom	Echo at 1 week		Total
	Normal	Congenital Heart disease	
No symptom	42 55.3%	34 44.7%	76 100.0%
Any symptom	4 18.2%	18 81.8%	22 100.0%
Total	46 46.9%	52 53.1%	98 100.0%

Symptom vs CHD at birth Chisquare p value – 0.002 (Significant)

Odds ratio for Symptomatic infants to have CHD at birth – 5.559

P value – 0.018 (significant)

95% confidence interval- 1.718 to 17.982 (significant)

Correlation between presence of symptoms and CHD at birth is significant.

Table 5B : Correlation between presence of symptoms and CHD at 6 weeks

Presence of symptoms	CHD at 6 weeks		Total
	NO	Yes	
No symptom	17 (53.1%)	15 (46.9%)	32 (100%)
Any symptom	1 (7.7%)	12 (92.3%)	13 (100%)
Total	18 (40.0%)	27 (60.0%)	45 (100%)

Symptom vs CHD at 6 weeks Chisquare p value – 0.005 (Significant)

Odds ratio for Symptomatic infants to have CHD at 6 weeks – 13.60

P value – 0.018 (significant)

95% confidence interval- 1.576 to 117.329 (significant)

Correlation between presence of symptoms at initial examination and persistent CHD at 6 weeks is significant.

Table 6 : Clinical features in study population

Clinical features	Number
Cyanosis	5
Low saturation	9
Edema	4
Respiratory distress	12
Bounding pulse	6

Table 6A : Correlation between presence of clinical features and diagnosis of CHD by Echo at 1st week of life			
Clinical features	Echo at 1st week		Total
	Normal	Congenital Heart disease	
No CF	42 (56.0%)	33 (44.0%)	75 (100%)
Any CF	4 (17.4%)	19 (82.6%)	23 (100%)
Total	46 (46.9%)	52 (53.1%)	98 (100%)

CF vs CHD at birth Chisquare p value – 0.001 (Significant)
Odds ratio for infants with CF to have CHD at birth – 6.045
P value – 0.003 (significant)
95% confidence interval- 1.875 to 19.491 (significant)

Correlation between presence of clinical features and CHD is significant.

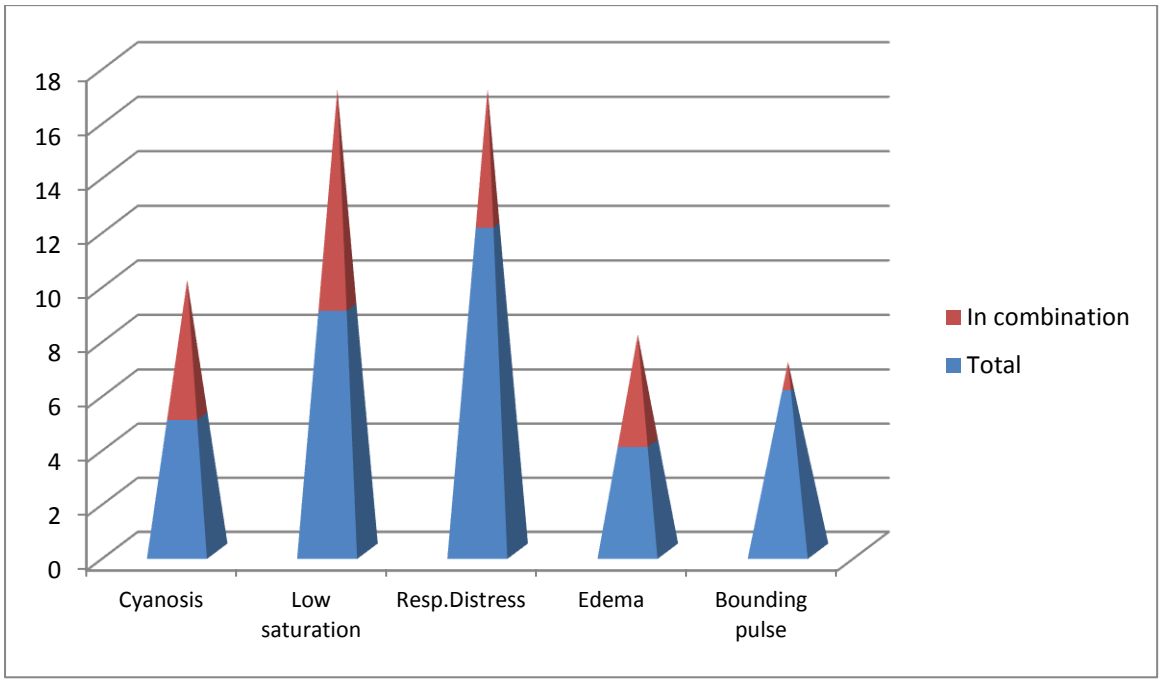
Table 6B : Correlation of Clinical features at birth with CHD at 6 weeks

Clinical features	CHD at 6 weeks		Total
	NO	Yes	
No CF	18 (58.1%)	13 (41.9%)	31 (100%)
Any CF	0 (0.0%)	14 (100%)	14 (100%)
Total	18 (40.0%)	27 (60.0%)	45 (100%)

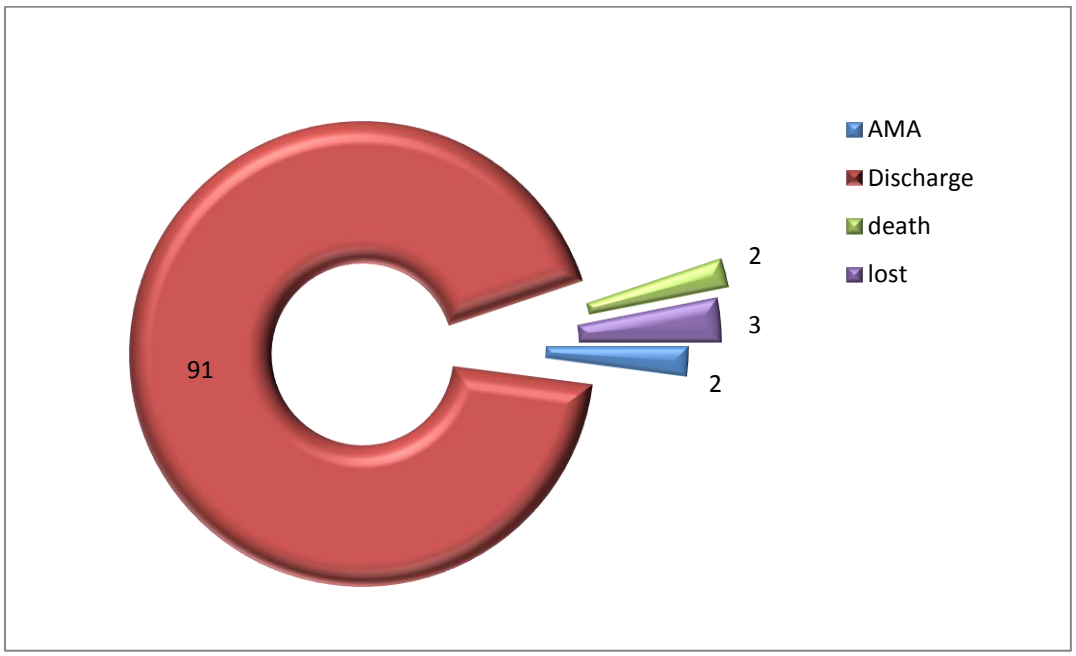
CF vs CHD at 6 weeks Chi-square p value < 0.001 (Significant)

Correlation between presence of clinical features at initial evaluation and persistent CHD is significant.

Graph 5: To show various clinical presentations



Graph 6: Outcome



Of 98 babies with murmur, 91 discarged. 2 babies were discharged against medical advice. 2 babies died during their hospital course. 3 babies did not turn up for follow up.

Table 7: Frequency of CHD based on Echocardiogram

ECHO DIAGNOSIS	Frequency	Percent
PHY PUL STENOSIS	2	2.0
ASD	9	9.2
ASD+PDA	3	3.1
ASD+TR	1	1.0
ASD+VSD	2	2.0
CYANOTIC	6	6.1
MILD TR	2	2.0
NORMAL	46	46.9
PDA	7	7.1
PFO	6	6.1
VSD	12	12.2
VSD+PDA	1	1.0
VSD+TR	1	1.0
TOTAL	98	100.0

GRAPH 7: Frequency of CHD

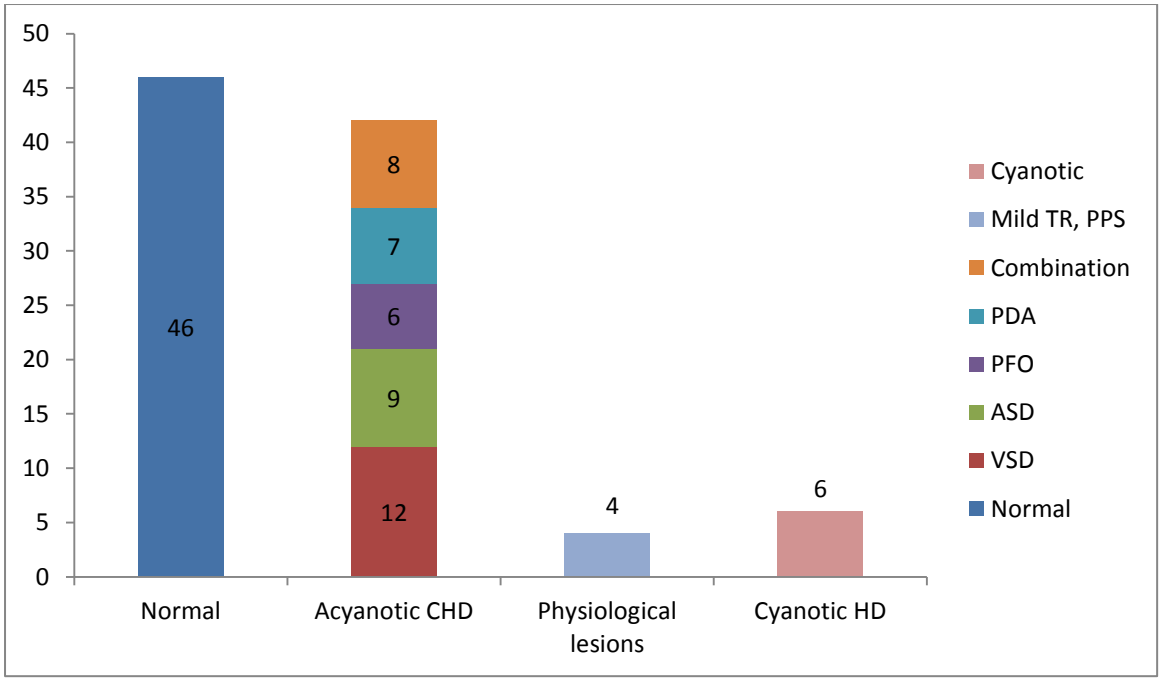


Table 8: Presence of Hepatomegaly

Hepatomegaly	Frequency	Percent
Absent	92	93.9
Present	6	6.1
Total	98	100.0

Table 9: Frequency of associated anomaly

Asso. Anomaly	Frequency	Percent
No	88	89.8
Absent radius	1	1.0
Anal atresia	1	1.0
Cleft lip	1	1.0
Diaph hernia	1	1.0
Down syndrome	2	2.0
Eso atresia,TEF	1	1.0
Polydactyly	1	1.0
Skin tag	2	2.0
Total	98	100.0

Table 10 : Association of murmur with thrill

Thrill	Number	Percent with respect to CHD
Present	36	69.2
Absent	16	30.8

Most of the cases with murmur having thrill were diagnosed to have CHD by echocardiography

Table 11: Day in which murmur is detected

Day	**Number (Percent)**
0 to 24 hrs	3 (3.06%)
24 to 48 hrs	14 (14.28%)
48 to 72 hrs	58 (59.18%)
>72 hrs	23 (23.46%)

Most of murmurs were detected on day 3.

Table 12: Presence of murmur at 6weeks in babies who had structural heart disease at initial evaluation

Murmur at 6weeks	Frequency	Percent
Not Applicable	53	54.1
NO	15	15.3
YES	30	30.6
Total	98	100.0

Of 45 babies followed up , 30 babies had persistent murmur.

Table 13: Presence of CHD at 6weeks

CHD at 6weeks	Frequency	Percent
Not Applicable	53	54.1
NO	18	18.4
YES	27	27.6
Total	98	100.0

Of 45 babies followed up, 27 had persistent congenital heart disease.

Table 14: Correlation between murmur at 6 weeks and structural heart disease

Murmur at 6weeks	HD at 6 weeks		Total
	NO	Yes	
NO	12(80%)	3 (20%)	15 (100%)
YES	6(20%)	24 (80%)	30 (100%)
Total	18(40%)	27 (60%)	45 (100%)

Murmur at 6 weeks vs. CHD at 6 weeks –chi square p value < 0.001 (significant)

Odds ratio for infants with murmur to have persistent CHD at 6 weeks – 16.0

P value < 0.001

(in comparison to infants with no murmur)

95% confidence interval- 3.398 to 75.345 (significant)

Correlation between murmur at 6 weeks and presence of CHD is significant

Table 15: Presence of X-ray abnormality

x-ray	Frequency	Percent
Negative	87	88.8
Positive	11	11.2
Total	98	100.0

Table 15A : Correlation between X ray abnormality and CHD at birth

X-ray abnormality	Echo at initial evaluation		Total
	Normal	Congenital Heart disease	
Absent	45 (51.7%)	42 (48.3%)	87 (100.0%)
Present	1 (9.1%)	10 (90.9%)	11 (100.0%)
Total	46 (46.9%)	52 (53.1%)	98 (100.0%)

X ray vs CHD at birth Chi-square p value – 0.008 (Significant)

Odds ratio for X-ray positive infants to have CHD at birth – 10.714

P value – 0.027 (significant)

95% confidence interval- 1.314 to 87.337 (significant)

Correlation between X ray abnormality and CHD at birth is significant.

Table 15B : Correlation between X ray abnormality and CHD at 6 weeks

X ray abnormality	Heart disease at 6 weeks		Total
	NO	Yes	
Negative	18 (48.6%)	19 (51.4%)	37 (100%)
Positive	0 (0.0%)	8 (100%)	8 (100%)
Total	18 (40.0%)	27 (60.0%)	45 (100%)

X-ray vs CHD at 6 weeks –chi square p value < 0.011 (significant)

Correlation between X ray abnormality and CHD at 6 weeks is significant.

Table 16: Presence of ECG abnormality

ECG	Frequency	Percent
Negative	88	89.8
Positive	10	10.2
Total	98	100.0

Table 16A : Correlation abnormality between ECG and CHD at birth

ECG abnormality			Echo at 1 st week		Total
			Normal	Congenital Heart disease	
ECG	Negative	Count	45	43	88
		(% within ecg)	(51.1%)	(48.9%)	(100.0%)
	Positive	Count	1	9	10
		(% within ecg)	(10.0%)	(90.0%)	(100.0%)
Total		Count	46	52	98
		(% within ecg)	(46.9%)	(53.1%)	100.0%

ECG vs CHD at birth Chisquare p value – 0.014 (Significant)

Odds ratio for ECG positive infants to have CHD at birth – 9.419

P value – 0.037 (significant)

95% confidence interval- 1.144 to 77.519 (significant)

Correlation between ECG abnormality and CHD at initial evaluation is significant

Table 16B : Correlation between positive ECG finding vs. heart disease at 6 weeks

ECG abnormality			CHD at 6 weeks		Total
			NO	Yes	
ECG	Negative	Count	17	21	38
		% within ecg	(44.7%)	**(55.3%)**	(100%)
	Positive	Count	1	6	7
		% within ecg	(14.3%)	**(85.7%)**	(100%)
Total		Count	18	27	45
		% within ecg	(40.0%)	(60%)	100.0%

ECG vs CHD at 6 weeks Chi-square p value – 0.131 (Not Significant)

Odds ratio for ECG positive infants to have CHD at 6 weeks – 4.857

P value – 0.161 (not significant)

95% confidence interval- 0.532 to 44.341 (not significant)
Hence ECG correlation with CHD at 6weeks is not significant.

Graph 8: To show the distribution observed variables positive at birth

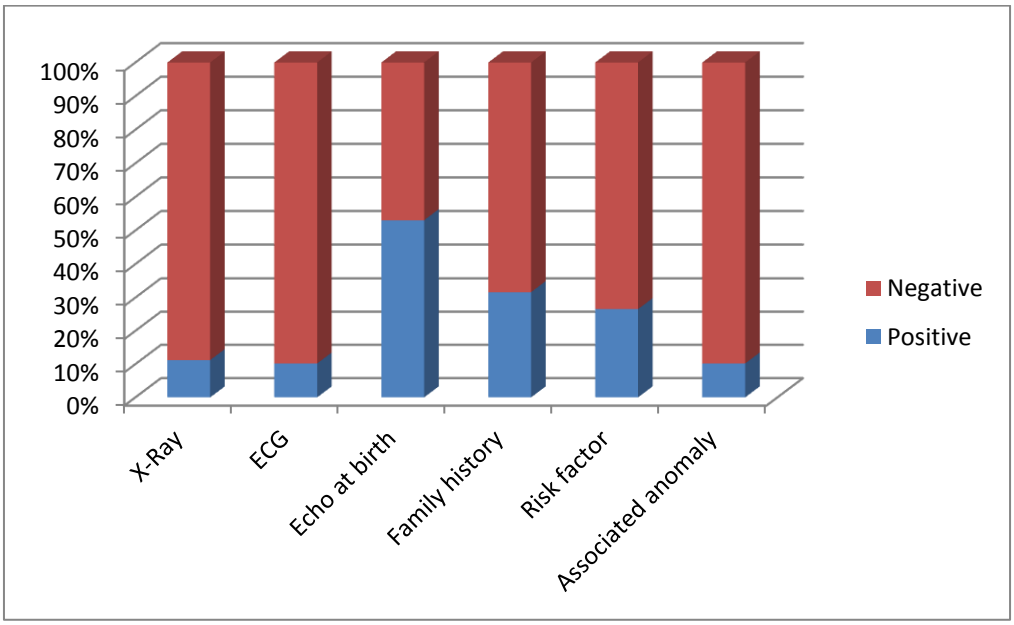


Table 17: Correlation between murmur diagnosis and echo diagnosis

Clinical diagnosis of murmur	Echocardiographic diagnosis	
	structural heart disease	Normal heart
Pathological	42	2
Innocent	10	44

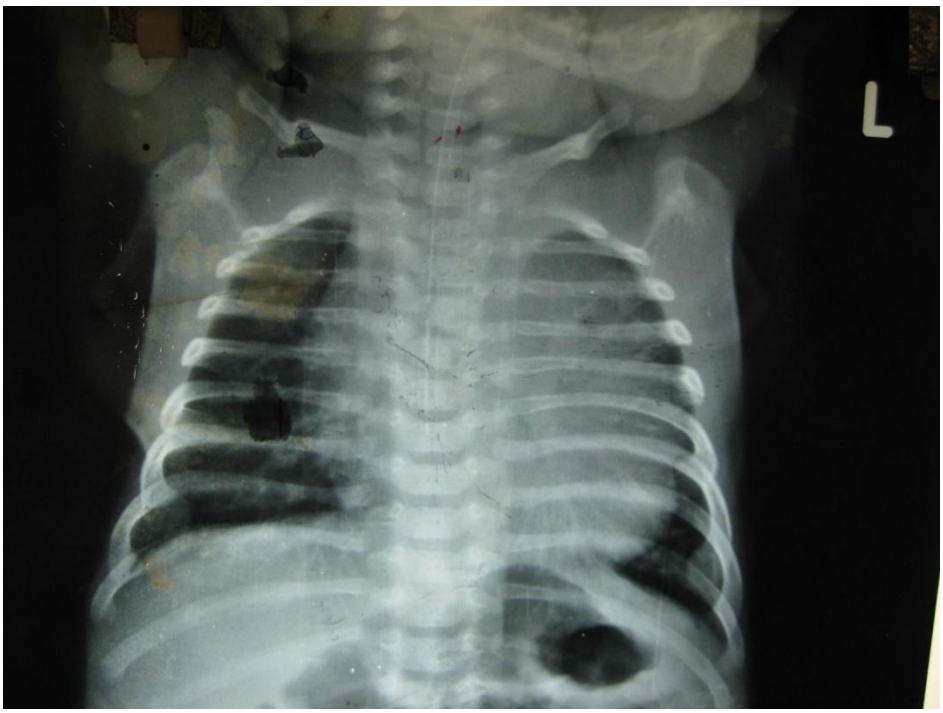
Odds ratio is 92.4 and p value < 0.001; confidence interval 19.10 to 446.80

When a murmur is pathological the chance of echo being positive is 92 times that of when the murmur is innocent.

Table 18: Comparison between various studies regarding incidence of congenital heart disease in neonates with murmur

STUDY	% of heart disease in neonates with murmur
Our study	53.06
Mehrdad Mirzharahmi et ai	51.60
Gregory R. Samson et al	75.00
Bansal M et al	45.00
laohprasitiporn	59.03
Du Z D et al	83.60
Amer Abdullah lardhi	42.50
Ainsworth et al	54.34
Mohammed Monu Hossain et al	68.00

CHEST X-RAY OF B/O INIYA – VSD WITH CARDIOMEGALY

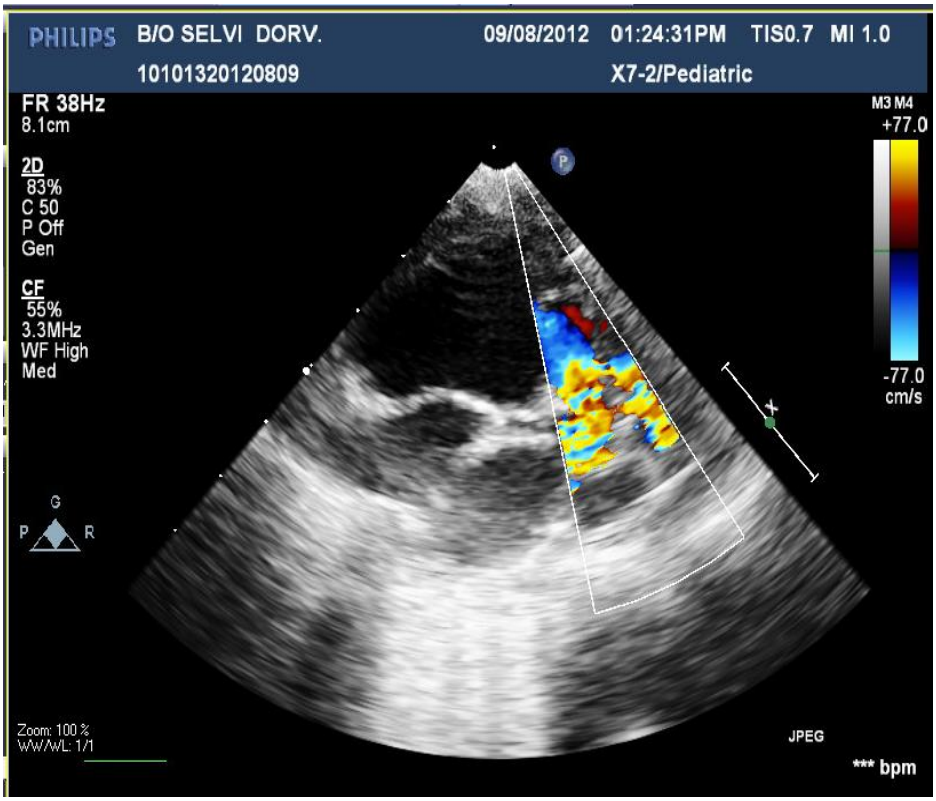


CHEST X-RAY OF B/O GEETHA WITH TGA

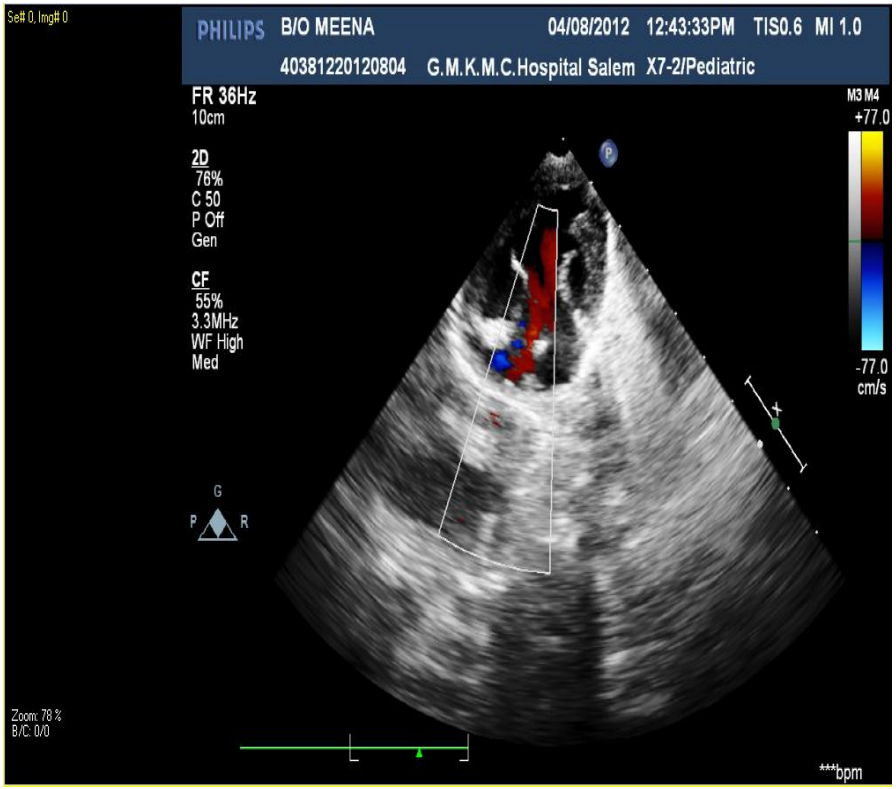
B/O MEENA- A CASE OF HETEROTAXY WITH SINGLE VENTRICLE

B/O RAJALAKSHMI- A CASE OF DOWN SYNDROME WITH ASD

ECHOCARDIOGRAM OF DOUBLE OUTLET RIGHT VENTRICLE



ECHOCARDIOGRAM OF SINGLE VENTRICLE



DISCUSSION

Congenital heart disease is one of the most common congenital malformations. Many present with this problem in neonatal period. Cardiac murmur is an important finding of CHD. There is a popular belief that heart murmurs are common in neonates and most of them are innocent. But a heart murmur may be a warning sign of serious CHD.

It is important to differentiate murmurs due to functional cause from structural cause. The earlier the CHD is diagnosed better is the prognosis. Therefore this study was done to evaluate the murmur in neonatal period.

In this study all term neonates with murmur were included. Detailed history is taken and clinical examination done. The clinical characteristics which differentiate pathological murmur from innocent murmur were noted. Chest X-ray and ECG were taken. It was later confirmed by echocardiography, which is the gold standard for diagnosis of CHD.

Total number of live births during the study period was 4116. Moribund babies who had murmur could not be evaluated completely. They were not taken up for study. Neonates with murmur who died before complete investigations were not taken up for study. 98 term

babies detected with murmur who can be evaluated completely were taken up for study. Incidence of cardiac murmur in our study was 23.8/1000 live births. Mehrdad Mirzharahimi MD et al[3] in their study had shown an incidence of 31/1000 live births having murmur. Bansal et al[5] in their study have shown 23.81/1000 live birth having murmur.

Of 98 babies 51 were female and 47 were male. Correlation between gender distribution and CHD at birth or at 6 weeks is insignificant.

In our study population 80 neonates were appropriate for gestational age. 3 were LGA and 15 were SGA. AGA is taken as reference category. There is no significant relationship between birth weight and CHD. This is similar to study done by Mehrdad Mirzharahimi MD et al[3].

Of 98 babies, there were no maternal risk factors for 72 neonates. 5 were infants of diabetic mothers. Mothers of 12 babies had polyhydramnios and 8 had oligohydramnios. 2 neonates were found to had intrauterine infection. Though the correlation between CHD and maternal risk factors was not significant, babies with maternal risk factors had two times more chance of having structural heart disease. This insignificance could be due to small sample size.

History of consanguinity was present in parents of 23 newborns (23.4%). History of CHD in family members is present in 11 newborns.

When there is history of consanguinity in parents or history of CHD in family members there is three times more chance of getting CHD. Correlation between family history and CHD is significant. (p =0.018). This is similar to studies by Becker S M et al [36] and Ramegowda et al [37] whose findings revealed increased incidence of CHD among babies of consanguineous parents.

Some of the neonates with murmur presented with symptoms of cyanosis, fast breathing, feeding difficulties and sweating. Fast breathing was most common symptom associated. 18 out of 52 babies with CHD presented with significant symptoms. 4 babies without CHD also had symptoms due to some other causes like sepsis, aspiration. Odds ratio for symptomatic neonates to have CHD is 5.559 and correlation was statistically significant (p=0.018).

Neonates with murmur also had clinical feature which assisted in the classification of murmur as innocent or pathological. Clinical features suggestive of CHD are respiratory distress (most common finding), cyanosis, low oxygen saturation, edema and bounding peripheral pulses. 19 out of 52 babies with CHD had clinical features at birth. At 6 weeks of follow up neonates with positive clinical features in addition to murmur had persistent structural heart disease. This correlation between clinical features and CHD at birth and at 6 weeks follow up was statistically

significant. E.Clarke et al[38] in their study suggested checking oxygen saturation in any neonate with suspicion of CHD. Cyanosis can be easily missed.

Some of the congenital anomalies, trisomies and dysmorphisms are seen in babies in the study. They are Down syndrome, neonate with absent radius, anal atresia, cleft lip, esophageal atresia with tracheoesophageal fistula, diaphragmatic hernia, polydactyly and skin tag. E.Clarke et al [38] suggested to consider a murmur as pathological when it is associated with dysmorphic features.

Down syndrome was most commonly associated with ASD in our study. A case of absent radius and thumb had ASD; this baby also had thrombocytopenia and turned out to be a case of *TAR syndrome.* A case with polydactyly had cyanotic heart disease. Cleft lip was associated with VSD. Another case of VSD had esophageal atresia with Tracheosaphageal fistula and was operated for the same. Baby withstood the procedure well and was discharged in good condition.

Hepatomegaly was present in 6 cases in the study group. Of them 4 were found to have structural heart disease. 2 other babies had liver enlargement due to septicaemia.

Chest X-ray showing cardiomegaly is taken as a positive finding. 10 out of 11 babies who had cardiomegaly were found to have structural heart disease. This correlation was statistically significant (p=0.008) at birth and also at 6 weeks. (p=0.011)

ECG abnormalities were found in 10 babies. Of them, 9 had structural heart disease. This correlation was statistically significant at birth (p=0.014) but was not significant at 6 weeks.

In the study of Swenson JM et al [39] chest X -ray and ECG helped in diagnosis of heart disease in 4 patients who were thought to have no heart disease. They recommended ECG and Chest X -ray as valuable tools for heart disease evaluation.

On Echocardiogram VSD was found to be the most common defect in 12 cases (23.1%). Ainsworth et al [12] in their study found that most common diagnosis was VSD (37%) followed by PDA (23%).

Next common defect was ASD in 9 cases (17.3%). Combination of acyanotic heart disease were found in 8 cases (3 cases had ASD with PDA, 2 cases ASD with VSD, remaining were VSD with PDA, ASD with TR and VSD with TR).

PDA was detected in 7 cases (13.5%). At 6 weeks follow up murmur disappeared in 3 babies. In 4 babies with persistent murmur 3 of

them had bounding peripheral pulse on initial examination. Two of them turned out to have persistent PDA at 6 weeks of life.

Six cases were found to have cyanotic heart disease (11.5%). In addition to murmur these babies had central cyanosis and low saturation in pulse oximeter. These clinical features aided in classifying the murmur as pathological. Cyanotic heart diseases observed in the study were

- 2 cases of TOF.
- Double outlet right ventricle with absent pulmonary valve
- Single ventricle with common AV canal defect (associated with midline liver and absent spleen- a case of *heterotaxy)*
- Transposition of great arteries.
- Dextrocardia with ventriculo arterial discordance with severe PHT

Some of the physiological variants found during the study are 2 cases of TR and 2 cases of physiological pulmonary branch stenosis. In these cases murmur and lesion both disappeared during the follow up.

Patent foramen ovale was present in 6 cases. These babies didn't have remarkable symptoms and signs, chest X-ray or ECG findings. Murmur disappeared in 4 cases at 6 weeks. Only one baby is found to have PFO at 6 weeks of time.

Of 98 babies with murmur, 4 cases were lost in the study. They were: 2 babies were discharged against medical advice. 1 baby with VSD had diaphragmatic hernia and was operated for the same. The baby was in neonatal ICU and died 3rd post op day. 1 baby with ASD with VSD had severe respiratory distress, shock and hepatomegaly. Inspite of respiratory and ionotropic support baby could not be revived. This baby had cardiomegaly in x-ray.

46 babies had normal heart in initial echocardiographic evaluation and they were not followed up at 6 weeks.

48 babies were discharged with counselling of parents regarding warning signs and the need for follow up at 6 weeks. 3 babies didn't turn up for follow up. They were cases of PFO, PDA and physiological pulmonary branch stenosis. Remaining 45 babies were examined for murmur at 6 weeks of life. Of them, 30 had persistent heart murmur. 24 (80%) out of 30 babies had structural heart disease on echocardiogram. 3 babies with structural heart disease did not have murmur. Correlation between murmur at 6 weeks and heart disease is significant. ($p<0.001$).

Based on clinical features, associated dysmorphism, positive chest x-ray and ECG finding murmur were classified as innocent or pathological. When a murmur is diagnosed clinically as pathological the

chance of getting structural heart disease in echocardiogram is higher and the correlation is found to be significant ($p<0.001$).

As the study involved only neonates with murmur 4 other congenital heart diseases without murmur which could have been missed if only murmur is looked for as evidence of CHD. A note of all these lesions had been made and they were as follows

- 2 cases of ASD
- 1 case of TGA
- 1 case of PDA

CONCLUSION

Every newborn born in this world deserves a careful and skilled first examination. It is imperative on the health care delivery system to provide this to all babies, as this is the first golden opportunity to detect congenital anomalies.

In our hospital based study the incidence of cardiac murmur is 23/1000 live births and if a murmur is detected there is a 53% chance of having structural heart disease. Among them 7.7% had severe form of cardiac disease. Hence it is important to evaluate all neonatal cardiac murmurs before they become symptomatic. Early diagnosis aids in early referral and early institution of appropriate therapy to reduce morbidity and mortality.

Clinical features, associated dysmorphism, chest x-ray and ECG findings aid in diagnosis of murmur as innocent or pathological. For lesion specific diagnoses Echocardiography is necessary.

By evaluation of these neonates based only on murmur few congenital heart diseases can be missed.

The importances of follow up at 6weeks are

i) The detection rate of CHD in cases with persistent murmur at 6 weeks is high.

ii) Most of the insignificant defects and physiologic variants disappear.

Hence it is mandatory to examine all the babies at follow up.

SUMMARY

- Incidence of murmur in the study was 23/1000 live births.
- Percentage of structural heart disease in neonates with murmur is 53.06% which is quite significant.
- VSD was the commonest defect. TOF was the commonest cyanotic heart disease detected.
- Physiological variants, insignificant PDA and PFO disappear by 6 weeks of life.
- Correlation between gender, birth weight and CHD is not significant.
- Correlation between maternal risk factor and CHD is not significant. This could be because of small sample size.
- Incidence of CHD is higher in neonates of consanguineous parents and when there is history of CHD in sibling.
- Incidence of persistent structural heart disease is higher when associated with clinical features like respiratory distress, cyanosis and low oxygen saturation.
- Babies with any other anomaly should be screened for CHD.

- Correlation between chest X ray abnormality and CHD is significant.
- Correlation between ECG abnormality and echocardiographic positivity is significant at initial evaluation but not at 6 weeks.
- The correlation between clinical diagnosis of pathological murmur and echocardiographic diagnosis of structural heart disease is significant.

RECOMMENDATION

In our country there is considerable number of deliveries taking place in Primary Health Centre and Subcentres. Village health nurses are trained to look for obvious external congenital anomalies. It will be prudent to train them in detecting cardiac murmurs so that the *golden opportunity* of early detection of CHD is not missed.

Prompt early referral for echocardiography is necessary for diagnosis and appropriate management. Structurally normal heart in echocardiography helps in authoritative re-assurance of parents.

LIMITATION

- Invasive studies like catheterization were not done.
- Autopsy was not done for babies who died before evaluation.
- Sample size and study duration is small. The follow up of babies after 6 weeks is not done.

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PROFORMA

CLINICAL AND ECHOCARDIOGRAPHIC EVALUATION OF NEONATAL CARDIAC MURMURS AND THEIR FOLLOW-UP AT 6 WEEKS OF AGE

CASE NO:

SL NO		
1.	NAME	
2.	AGE	
3.	SEX	
4.	IP NO	
5.	FATHER'S NAME	
6.	ADDRESS	
7.	PHONE NO	
8.	DATE & TIME OF BIRTH	
9.	BIRTH WEIGHT	
10.	BIRTH ORDER	
11.	MODE OF DELIVERY	
12.	PLACE OF BIRTH	
13.	APGAR 1MIN & 5 MIN	
14.	H/O CONSANGUINITY	
15.	MATERNAL MEDICATIONS	
16.	MATERNAL INFECTIONS	
17.	MATERNAL SMOKING	
18.	MATERNAL ALCHOHOLISM	
19.	MATERNAL DIABETES	
20.	SIBLING H/O CHD	
21.	FAMILY H/O CHD	
22.	C/O FAST BREATHING	
23.	C/O FEEDING DIFFICULTY	
24.	C/O CYANOSIS	
25.	C/O SWEATING	
26.	TERM/POST TERM	
27.	AGA/SGA/LGA	
28.	PALLOR	
29.	CYANOSIS	
30.	PUFFY EYELIDS	
31.	ABNORMAL FACIES	
32.	FEAUTURES OF CHROMOSOMAL ABNORMALITY	
33.	OTHER MALFORMATIONS (CLEFT LIP/PALATE, POLYDACTYLY, SKIN TAGS, ETC)	

34.	TEMP	
35.	HR	
36.	PULSES IN ALL 4 LIMBS	
37.	RR	
38.	BP IN ALL 4 LIMBS	
39.	SAO2 IN ALL 4 LIMBS	
40.	PRECORDIAL ACTIVITY	
41.	APICAL IMPULSE	
42.	THRILL	
43.	CVS AUSCULTATION S1 & S2 OTHER SOUNDS	
44.	MURMUR SYSTOLIC/DIASTOLIC SITE GRADE	
45.	RS EXAMINATION	
46.	ABDOMEN EXAMINATION HEPATOMEGALY SPLENOMEGALY ASCITES OTHERS	
47.	CNS EXAMINATION	
48.	ECG FINDINGS	
49.	CHEST X-RAY FINDINGS	
50.	ECHOCARDIOGRAM FINDINGS	
51.	DIAGNOSIS	
52.	FOLLOWUP AT 6 WEEKS PRESENCE OR ABSCENCE OF MURMUR ECHOCARDIOGRAM	

CONSENT FORM

I _____ in my full senses hereby give complete consent for investigations and interventions performed on my baby / ward. For academic and scientific purpose , the results can be photographed

Date

Signature / Thumb impression of

the parent / Guardian

Name & Address